THE

HEALTH EFFECTS

OF

WELDING AND CUTTING FUME

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EXECUTIVE SUMMARY

The numerous welding and cutting processes, the variety of base metals and types of consumables (e.g. welding rods) combine to form a complex array of many different exposure environments. Fusion welding, in which metals are joined by heat, includes gas and electric arc processes; the latter is the most common welding technique and has received the most study for health effects. It has been estimated (Stern, 1983a) that two major arc welding technologies [shielded metal arc welding (SMAW) with stick electrodes; and gas metal arc welding (GMAW) with continuous wire] applied to mild steel, stainless steel or aluminum, account for combinations practised by 70 per cent of welders. The Hatch Report (1985) suggested that these estimates are also applicable to the Ontario situation. The welding process produces potential respiratory hazards inherent in the welding process itself, including a particulate fraction, which is largely respirable metal oxide fume, and gases, of which ozone and oxides of nitrogen are most dangerous. The welding environment may also contain contaminants that are, strictly speaking, extraneous to the welding process, such as gases (for example, phosgene formed from decomposition of chlorinated hydrocarbons remaining on metal surfaces after degreasing), and particulates such as those resulting from surface paint or from bystander exposure to asbestos or silica.

Certain welding techniques used on particular metals may produce process-specific hazardous exposures: for example during SMAW, and to a lesser extent, GMAW, of stainless steel, exposure to hexavalent chromium may occur; during GMAW with aluminum or stainless steel excessive ozone exposure may occur. Also high exposure to nitrogen dioxide may occur in confined spaces and potentially toxic exposures can be associated with welding with specific consumables or on specific coatings. As a generalization, (but with those few exceptions just described), exposures to constituents do not generally exceed individual threshold limit values or

similar criteria, if total fume is kept to about 5 mg/m^3 (Stern, 1981a).

Studies have shown that urinary (and blood) levels of chromium and nickel are increased in stainless steel welders, as are the urinary aluminum concentrations of aluminum welders. Pathological investigations, and more recently, magnetopneumography, have demonstrated that pulmonary dust burdens in welders are increased relative to non-welders. The latter technique has been used to estimate the amount of lung-retained metal contaminants; average clearance for mild steel dust was estimated to be 20 per cent per year (Kalliomaki et al.. 1983d).

Investigations in rats indicate that there may be differences in pulmonary clearance between fume types; for example, chromium from GMA/stainless steel fume was cleared slowly (half-time 240 days) but was cleared rapidly from SMA/stainless steel fume (half-time 40 days).

Acute, welding-associated health effects in humans consist mainly of case reports related to the upper and lower respiratory tract system. Such intoxications have resulted in acute pneumonitis, bronchitis and pulmonary edema including fatalities associated with exposure to ozone, oxides of nitrogen, phosgene and cadmium fume. These exposures are largely process-dependent as described above, occur in confined spaces, or are due to a lack of awareness of the composition of consumables or coatings. At lower concentrations, eye and throat irritation may be due to, for example, ozone or fluorides. There have been reports of occupational asthma with stainless steel welders, probably due to chromium or nickel. Metal fume fever (MFF) is an acute febrile illness of short duration that affects up to 30 per cent of welders and results from inhalation of freshly formed metal oxides, most commonly that of zinc. The cause is not known but the syndrome resolves leaving no apparent chronic disorder. Although MFF occurs below the threshold of 15 mg/m^3 proposed by Drinker et al., (1927),

data are insufficient to confirm the occurrence of attacks below 5 mg/m^3. Also, in comparison with their wives, welders have been found to suffer from excess mortality due to pneumonia. It is possible that some deaths were due to acute exposures that were not recognized as such.

Because of the multitude of processes, confounding exposures (e.g. cigarette smoking) extraneous to the welding process itself, poor study design and lack of environmental data, the existence and magnitude of the burden of chronic disease in welders have not been easy to determine. Thus, the focus has continued to be on the respiratory tract. Welders do appear to exhibit more absenteeism attributed to respiratory (especially lower respiratory) tract disease compared to non-welders (McMillan, 1981; Fawer et al., 1982). These epidemiologic studies which assess chronic respiratory effects in welders (Appendix to Part II) are all cross-sectional in design (and thus suffer from the problems inherent in this study design); most of these studies are concerned with mild steel welders. Nevertheless, there appears to be a consistent increased prevalence, compared to the comparison groups employed. of chronic pulmonary symptoms (particularly sputum production and cough) in both smokers and non-smokers, and in both shipyard and non-shipyard welders. However, there is not sufficient data with which to relate this finding to duration of exposure, to a specific constituent within welding fume or to a particular level of such a constituent. There is a suggestion, also determined from cross-sectional studies, of a difference in lung function between welders and available controls, indicating an excess in welders of mild obstructive airways disease. Longitudinal prospective studies will be necessary to determine the incidence of such abnormalities and to clarify the rate of decline with time.

Siderosis, a pneumoconiosis due to pulmonary deposition of iron particles, appears as nodular radiopacities on chest x-ray. Siderosis itself is considered to be benign, without fibrosis, and if fibrosis is present, is considered to be

due to exposure to other dusts, such as crystalline silica (Parkes, 1982). The prevalence of siderosis has been shown to increase with duration of exposure (Attfield and Ross, 1978). However, no studies have been reported which would permit a correlation between exposure level to iron oxide and the development of x-ray changes. Despite the apparently benign nature of siderosis, there are a number of case reports of symptomatic pulmonary disease accompanied by fibrosis and emphysema; these cases were documented by lung biopsy or autopsy (Stern et al., 1983). Morgan and Kerr (1963) attributed these effects to mixed dust exposure while Stern et al., (1983) have suggested that the fibrogenic potential of welding fume may involve exposure to hexavalent chromium or NO_2. Investigations of other chromium-exposed working populations, however, provide little evidence to suggest a role for chromium in the production of chronic non-malignant respiratory disease. Although NO_2 is an experimental fibrogen, Parkes (1982) considered that there was no adequate proof that intermittent exposure to NO_2 causes emphysema in humans. Moreover, as there is no estimate of the incidence of fibrosis in welders (only case reports), it is not possible to demonstrate that an excess incidence in welders does, in fact, exist.

Traditionally, the search for effects associated with exposure to welding fume and gases has focused on chronic respiratory diseases, while non-respiratory effects have been neglected (Zielhuis and Wanders, 1985). Consequently, the reason for the apparent lack of associations of welding with non-respiratory effects may be due to a lack of appropriate investigations. Chronic neurologic disorders attributed to manganese exposure have been rarely described. Contact dermatitis with patch tests results demonstrating a sensitivity to chromates has been reported in welders but it could not be demonstrated that the welding exposure was responsible for the sensitization.

Stainless steel, but not mild steel, welding fume appears to be mutagenic in in vitro tests in bacteria and in cultured mammalian cells. The mutagenic effect appeared to be associated with the water soluble fraction and hexavalent chromium has been considered as one of the agents responsible for this activity. The only two studies reported have failed to detect chromosomal changes in lymphocytes of stainless steel welders but these studies did not exclude the possibility of point mutations.

The welding environment contains a number of known or suspected metal carcinogens, two of which, nickel and chromium, appear in significant concentrations in stainless and some alloy steels. In addition, pyrolytic decomposition products from paints used on mild steel contain a mixture of organic materials of unknown carcinogenicity, and bystander exposure to carcinogens, such as asbestos, may occur.

Although some studies (mostly case-control studies) have suggested welders have an increased risk of some non-respiratory cancers, further work is needed to confirm these findings.

Attention has been drawn to assessing the possible risk of lung cancer in welders because of the presence in welding fume of metals of known or suspected carcinogenicity in other settings, the respirable nature of the fume, and the route of entry by inhalation. There is, however, no experimental evidence documenting the carcinogenicity of welding fume. Studies based on death certificates and/or census returns, on shipyard welders, and on non-shipyard welders are consistent with a relative risk for lung cancer of the order of 1.3 or 1.4 compared with referent groups, usually national mortality rates (Peto, 1985). Investigations in England and Wales (Registrar General, 1978) but not in Sweden (Sjogren, 1980) have suggested that welders may smoke somewhat more than the general male population. This increased lung cancer risk may be due to the confounding effects of smoking (and perhaps exposure to

other occupational carcinogens). Alternatively, mild steel welding may involve little or no risk but certain welding processes, in particular stainless steel, may constitute a "hot spot" (Stern 1983a,b) with a higher risk of lung cancer. This explanation considers that stainless steel welding fume contains hexavalent chromium, that stainless steel welders absorb chromium, that stainless steel welding fumes contain mutagenic compounds, that chromium (VI) appears to be the (an) active contributor to this mutagenicity, and that there are recognized increased relative risks for lung cancer in other industries where workers are exposed to chromates. The only published evidence of an increased lung cancer risk in stainless steel welders is the study by Sjogren (1980) in which a 4-fold relative risk was reported but this study was very small (based on only three cases) and needs to be confirmed. Any risk from nickel appears negligible compared to that from chromium (VI) (Stern, 1983a).

The third possible explanation for a slightly increased risk of lung cancer in welders is that long-term employment as a welder of any type may cause an increase in lung cancer after a sufficient latency period has elapsed (for example, 20 years), as demonstrated by Beaumont and Weiss (1981). This was one of the only studies to include an analysis of findings by duration of exposure and latency. No relationship with duration of exposure was found in this study. There is, as yet, no candidate carcinogen identified in mild steel fume, and mild steel fume has not been shown to be mutagenic.

The available epidemiologic studies of lung cancer in welders generally suffer from methodologic limitations (small population size, confounding exposures, mixed processes, absence of quantification of exposure data, absence of analysis by duration of exposure or by latency). To decide whether the association between lung cancer and welding is a causal one, large cohorts of welders consisting of groups with long exposure to stainless steel

and mild steel who have been followed for more than 20 years from their first exposure to welding, must be assembled. The existing studies do not meet these criteria.

Taken as a whole, there is reason for concern that prolonged exposure to excessive concentrations of welding fume under past and possibly present conditions may present some degree of hazard to the health of welders. But the data do not permit the health effects to be related to cumulative duration of exposure, much less to levels of specific constituents which can be deemed safe or not safe, as they relate specifically to the welding environment.

Combined with the above discussion, there are known exposures of welders to total fume and to specific constituents in excess of current guidelines in Ontario and elsewhere (Jenkins et al., 1981). These include the process-dependent exposures (such as chromium and ozone) emphasized before. Thus, it would appear that exposure to welding fume should be controlled, whether by adjustments directed at parameters of the welding process, by recommendations for ventilation, by an exposure guideline for total welding fume, or by a combination of all of these approaches.

GLOSSARY

Welding is a process for joining metals rendered liquid by heating, by pressure, or both. Fusion welding, which includes gas and electric arc welding, involves heating the metals to a suitable temperature. In arc welding, the metal temperature is around 4000°C when workpieces fuse together, although the arc temperature is much higher. The various welding processes will not be described here. However, Stern (1983a) has pointed out that two major technologies (shielded metal arc welding using stick electrodes; and gas metal arc welding using continuous wire) applied to mild steel, stainless steel and aluminum account for combinations practised by 70% of welders.

Cutting or scarfing (which can be classified as a type of welding and which may have similar health effects), may involve cutting slab steel, removing gates and risers on castings. or gouging out defective metal in castings. Gates are the end of the runner in a mold where molten metal enters the casting or mold cavity. Risers are the reservoirs of molten metal provided to compensate for the contraction of the casting as it solidifies. In gouging, a strip of surface metal is removed but the metal is not cut through. Carbon arc cutting is an arc cutting process in which metals are severed by melting them with the heat of an arc between a carbon electrode and the base metal. In oxygas cutting, the metal is heated by a flame and a jet of pure oxygen is directed to the point of cutting, and moved along the line to be cut. Cutting differs from welding in that only one piece of metal may be involved and temperatures are somewhat lower than in welding.

Burning is the removal of surface materials, especially paint and is also improperly used to describe the process of cutting. Lead welding, often called "lead burning", is the fusion welding of lead. The heat required for lead welding is much lower than that for welding cast iron and steel.

Brazing can be defined as a technique for joining metals using a filler metal with melting point greater than 427°C (800°F) and is thus differentiated from soldering which uses filler metal or solder with a melting point less than 427°C. The processes differ from arc welding in that the filler metal is flame or contact melted rather than melted by an electric arc.

Abbreviations for Processes

Process	Abbreviations	Synonym	Abbreviations
Flux cored arc welding	FCAW		
Gas metal arc welding	GMAW	Metal inert gas	MIG
Gas tungsten arc welding	GTAW	Tungsten inert gas	TIG
Shielded metal arc welding	SMAW	Manual metal arc ("stick")	MMA
Shielded metal arc welding of stainless steel	SMA/SS		
Shielded metal arc welding of mild steel	SMA/MS		
Gas metal arc welding of stainless steel	GMA/SS		
Gas metal arc welding of mild steel	GMA/MS		

INTRODUCTION

This document reviews the health effects associated with occupational exposure to welding and cutting fumé. The effects of associated physical agents including: noise, ergonomic, and musculoskeletal problems, burns, eye injuries ("arc eye"), ultraviolet radiation, and electric shock are not considered but have been reviewed in detail elsewhere (Villaume et al. 1979; Zakhari et al. 1983; Palmer, 1983; Tinkler and Graville, 1983; Bell, 1975).

The complexity of the problem cannot be overemphasized, both in terms of the variety of processes and the spectrum of exposures. In addition, the exposure received by the welder varies with other factors including the site of the welding operation, the work practices of the welder, the use of protective devices, and fume suppression (Newhouse and Murray, 1981). From 0.2 to 2.0 per cent of the working population in industrialized countries has been estimated to be engaged in welding (Stern, 1981a). In Ontario, it has been estimated that up to 36,000 residents were employed as welders (Hatch Associates, 1985).

Welding is a process of fusing metals together by melting. Electric-arc welding, the major type, uses various techniques to protect the melting metal from the oxidizing power of the air. For example, the melted metal can be protected by material in the coating of the electrode (as in shielded metal arc welding) or by an inert gas (as in gas-shielded welding). Gas-shielded welding can be of two types: gas metal arc welding or metal inert-gas welding which uses a continuous wire consumable electrode, and gas tungsten arc welding or tungsten inert-gas welding which uses a nonconsumable tungsten electrode. Further definitions of welding and the related processes of cutting and burning, plus abbreviations used in this report are presented in the Glossary.

The technical aspects of the numerous welding and cutting processes have been summarized in the report by Hatch Associates (1985: Part 1, Section 2) and are not described here. The reader is also referred to Hatch Associates (1985) for a description of welding and cutting processes used in Ontario (Part 2, Section 2.2): for a summary of Workers' Compensation claims for welders in Ontario (Part 2, Section 4); for a review of variables influencing exposure (Part 1, Section 4.0) and for the Ontario experience with respect to levels of exposure to welding fume and its constituents. This includes summaries of air quality assessments performed by the Ministry of Labour (Part 2, Section 3) and of a survey of a number of welding and cutting establishments performed on contract for Hatch Associates (Part 2, Section 5) as part of its review of welding hazards in Ontario.

There is uncertainty in the determination of both the chronic effects of individual fume constituents and their safe exposure levels even before starting to consider all the components with their possible interactions. Other exposures that may be important include those considered of occupational origin but extraneous to true welding fume (e.g. asbestos) and those of non-occupational origin (e.g. cigarette smoking). Such interactions can be synergistic in nature (e.g. asbestos and smoking in the production of lung cancer): also, efforts to control or decrease one aspect (eg. welding fume) can be associated with an increase in another (eg. ozone levels). Moreover, even where the effects of important confounding factors are known (such as cigarette smoking in the assessment of pulmonary function), some studies of the health of welders have not adequately controlled or did not even address these factors. Finally, as in occupational health studies in general, exposure data may be inadequate or nonexistent.

Stern (1981a) differentiated between the generalized risks of welding as indicated by reports of metal fume fever, or of accidental death due to electric shock, and risks which were process-dependent. An example of the latter is exposure to water soluble hexavalent chromium associated with welding of stainless steel in confined spaces.

The document is assembled in two portions. In the first, summaries of the known potential health effects for the more important welding fume constituents are presented, without examining the extent to which effects have actually been documented in welders. These summaries are accompanied by a description of the principal welding processes where one would expect these contaminants to be encountered. In the second part of the document, health effects documented in welders are reviewed taking into account, where possible, certain methodological criteria for epidemiological investigation (ACOHOS, 1983).

PART I

SUMMARIES OF SPECIFIC CONSTITUENTS

The emphasis in the literature pertaining to welding and cutting deals with the health effects of welding (and within welding, with arc welding). The possible constituents or components of welding fume categorized according to their broad health effects, are displayed in Figure 1 (American Welding Society, 1973). It is not the purpose of this document to summarize the health effects of all of these constituents. They have been assigned individual specific guidelines or recommendations for limiting exposures, such as Threshold Limit Values (TLVs) recommended by the American Conference of Governmental Industrial Hygienists (ACGIH, 1984).

One can think in terms of where one might expect to encounter such constituents (Table 1; Burgess, 1981). Rather than merely tabulating potential health effects of all contaminants, brief reviews of the known health effects of certain major constituents follow, emphasizing those considered of high and intermediate priority (Tinkler and Graville, 1983; see Table 1). These reviews are followed by a notation of those welding processes where one would expect these constituents to be encountered. Approximate ranges of the exposure levels of constituents commonly found in welding fume are summarized in Appendix I.

1.0 Gases

Gases encountered during welding may be intrinsic to the welding process. Examples of these include atmospheric gases (such as ozone and oxides of nitrogen), or shielding gases (such as argon and CO_2). On the other hand, gases may arise as by-products of the welding process due to coatings and contaminants on the surfaces being joined (such as phosgene formed from the decomposition of chlorinated

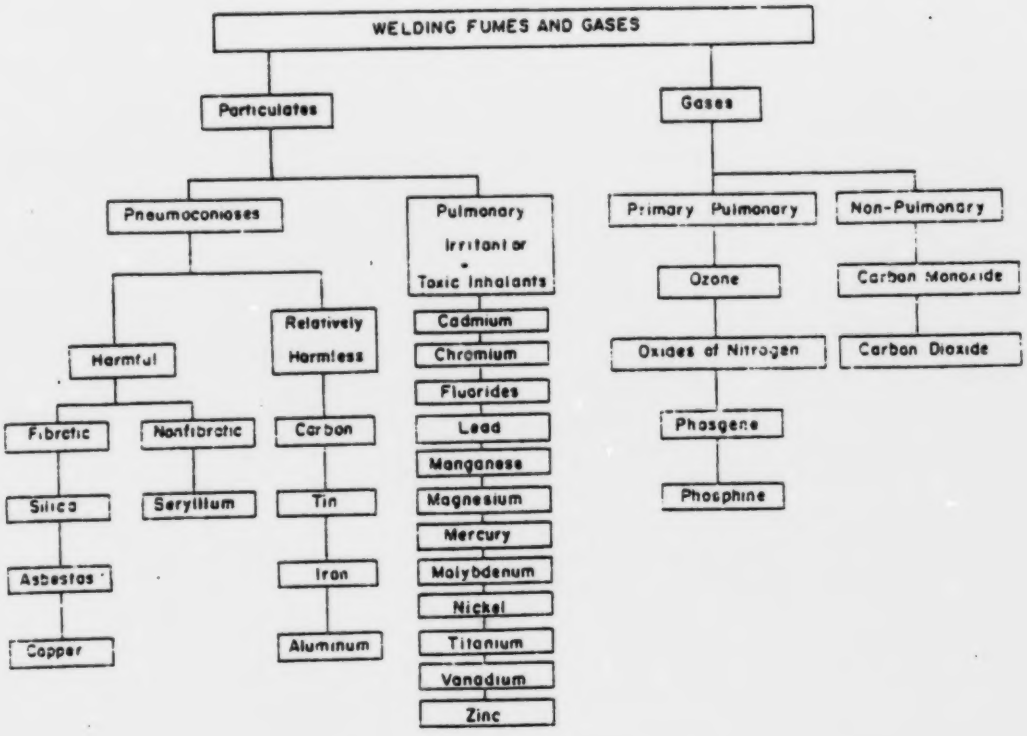

Fig. 1 - Possible constituents of welding fumes.

(from American Welding Society, 1973)

hydrocarbon solvents in air or on metal) and thus extraneous to the welding process itself. Finally, in addition to possible toxic effects, it is possible for oxygen depletion to occur in a confined space due to a build up of shielding gas.

1.1 Ozone (O_3)

Ozone is a powerful oxidizing agent. Its toxicity is believed to be related to oxidation of thiols or amino acids in tissue proteins or low molecular weight peptides and/or oxidation of unsaturated fatty acids with resultant interference with cell function and possible cell death. Most of the available data relates to its effects on the respiratory system but there is some evidence to suggest extrapulmonary effects.

Animal Studies: Inhalation of ozone at about 3-4 ppm for about 3-4 hours produces (pulmonary) edema (National Academy of Sciences, 1977). At ozone exposure concentrations of less than 1 ppm for variable durations, the junction of the conducting airways and the alveoli is the most sensitive area of the respiratory tract; at higher concentrations damage is more severe and widespread. Type I pneumocytes are usually damaged with hyperplasia of Type II pneumocytes. Several studies report interstitial thickening of the intraalveolar septa of alveoli following exposure to 0.5 - 1 ppm ozone over several weeks. This is considered related to the development of mild interstitial fibrosis (Moore and Schwartz, 1981; Last et al. 1979; Boorman et al. 1980; Freeman et al. 1973). Increased collagen synthesis and propylhydroxylase activity has been reported at exposure concentrations of 0.5 ppm and above (Last et al. 1979; Hesterberg and Last, 1981). These results would suggest a relationship between exposure and the development of fibrotic lung disease; however, there is no evidence that this occurs. In fact the above changes may decrease with prolonged exposure.

Studies on pulmonary function in animals report the onset of rapid shallow respiration following short-term (2 hours) exposure to concentrations of 0.2 - 0.5 ppm which disappears rapidly after exposure has ceased (Amdur et al. 1978). Longer lasting effects on residual volume, closing capacity and closing volume have been observed (Inoue et al. 1979). Longer term exposure (4-6 weeks) to 0.2 - 0.25 ppm and 0.4 - 0.8 ppm resulted in increased lung distensibility in young and old rats respectively (Bartlett et al. 1974; U.S. E.P.A., 1984; Moore and Schwartz, 1981). Increased pulmonary resistance was observed in rats and monkeys exposed to 0.6 and 0.8 ppm for 3-12 months; lung distensibility was decreased in the post-exposure period in monkeys (Wegner, 1982).

Tolerance to ozone is achieved by a few days of exposure; cessation of exposure leads to recovery of sensitivity. Animal experiments have demonstrated increased susceptibility to respiratory infections following ozone exposure.

Ozone is radiomimetic and extra pulmonary effects with chronic exposure including premature aging have been reported (ACGIH, 1980). The TLV of 0.1 ppm represents a limit which, although it results in no injury, "may result in premature aging if exposure is sufficiently prolonged" (ACGIH, 1980).

Human Studies: Controlled studies which evaluate the effect of ozone on pulmonary function in humans have been conducted under various conditions of exposure - at different exposure concentrations, different degrees of exercise, and in different exposure groups. In general, effects on flow rates, airway resistance, the incidence of symptoms and ability to do work during exposure are the parameters measured. Healthy adults exposed at rest demonstrated decrements in forced expiratory flow and volume when exposed to concentrations of greater than or equal to 0.5 ppm for a period of 2 to 4 hours (U.S. E.P.A., 1984; Horvath et al.

1979). Exercising healthy males exposed for 1-3 hours showed significant decrements in pulmonary function (usually expiratory flow rates and volumes), at even lower concentrations. The effect level was related more to the degree of exercise than to the type of exposure (i.e. intermittent or continuous). Effects were seen at or above 0.37 ppm under conditions of light, intermittent exercise; at or above 0.3 ppm with moderate exercise; at or above 0.25 ppm with heavy exercise; at or above 0.18 ppm and possibly as low as 0.12 ppm with very heavy exercise (U.S. E.P.A., 1984).

The significance of these experimentally observed decrements in pulmonary function is difficult to evaluate since such changes would clinically be considered insignificant. The presence of such decrements in population groups raises the possibility of a contribution to long-term effects, but to date there is insufficient data to evaluate this possibility.

Subjects with asthma or chronic obstructive lung disease do not appear to be particularly sensitive to ozone concentrations of 0.2 - 0.3 ppm even when combined with light exercise (Linn et al. 1978; Silverman, 1979; Linn et al. 1983; Solic et al. 1982).

Tolerance observed in animals is also seen in humans. Effects are greatest on the second day of exposure and usually disappear by the fifth or sixth day of exposure. Tolerance is lost after a period of non-exposure.

Epidemiological studies suggest that acute exposure to 0.08 - 0.15 ppm can cause altered pulmonary function (peak flow and airway resistance) in children (U.S. E.P.A.,1984). Results of studies in asthmatics and groups with chronic obstructive pulmonary disease are difficult to interpret because of the influence of medication on the parameters studied. Eye irritation is likely to occur at oxidant levels of about 0.10 ppm. Associations between oxidant

levels and symptoms such as eye, nose and throat irritation, chest discomfort, cough and headache have been reported above 0.1 ppm in both children and young adults (U.S. EPA., 1984). There would appear to be an association between the onset of these symptoms and ozone concentrations of 0.08 ppm in adults with asthma and allergies (Zagraniski, 1979). Others have reported effects under conditions of exercise at concentrations of 0.12 ppm and above.

Studies which evaluate the long-term effects of low level ozone exposure are not available. Studies which attempted to correlate ozone exposure with mortality often showed a closer association with temperature than with ozone concentration.

Welding processes where encountered: During welding, ozone is produced by the photochemical action of ultraviolet (UV) radiation on atmospheric oxygen, as illustrated below (Villaume et al. 1979):

$$O_2 \xrightarrow[\text{radiation } <210 \text{ nm}]{\text{ultraviolet}} 2\,O$$

$$O + O_2 \longrightarrow O_3$$

Ozone can be encountered in potentially hazardous concentrations in gas metal arc welding (GMAW), gas tungsten arc welding (GTAW) and arc air gouging with the highest concentrations recorded during the GMA welding of aluminum. Within this latter category, potentially high ozone exposure may occur during the bare-wire argon-shielded welding of aluminium, aluminium alloys and stainless steel (Lunau, 1967). Ozone production may be related to the effect of the shielding gas increasing the intensity of the UV radiation and to the reflectivity of the base metal (Bell, 1975). Approximate exposure ranges are found in Appendix I.

1.2 Nitrogen Oxides

Oxides of nitrogen include nitrous oxide (N_2O) used as an

anaesthetic gas, nitric oxide (NO) and the nitrogen dioxides (NO_2 which is dark brown, and N_2O_4 which is colourless). Nitric oxide and nitrogen dioxide are formed when nitric acid reacts with reducing agents (organic matter) as well as by combustion of nitrogen-containing materials. The effects due solely to NO_2 may be difficult to distinguish from those due to other gaseous components of welding fume, such as ozone, as they may have similar actions in the lung. Nitric oxide is converted spontaneously in air to nitrogen dioxide.

Mechanism of action: Unlike irritants affecting chiefly the upper respiratory tract, nitrogen dioxides are of lower water solubility and reach the terminal respiratory passages or peripheral airways. NO_2 undergoes hydrolysis to nitric acid which dissociates in the lungs into nitrates and nitrites resulting in local tissue damage, and including chemical pneumonitis leading to pulmonary edema. Following exposure to high concentrations of NO_2, methemoglobinemia can occur (Parkes, 1982). Nitrogen dioxide is a powerful oxidant, and can produce lipid peroxidation. NO_2 also acts to reduce host resistance to bacterial and viral respiratory infections, demonstrated by increased mortality following challenge with bacteria at levels above 1 or 2 ppm (summarized by Dawson and Schenker, 1979). Interference with macrophage activity appeared to be involved.

Acute effects from high concentrations: The acute effects resulting from exposure to high concentrations are known as a result of (usually accidental) situations such as the 1929 Cleveland Clinic disaster which took the lives of many patients and physicians following the burning of nitrocellulose x-ray films which liberated clouds of NO_x (Guidotti, 1978). Also NO_2 can be released from silage, producing silo-filler's disease in farmers on entry to silos that had not been vented.

Following high exposures, there may be no upper airway irritation and respiratory symptoms may be delayed 1-24 hours. Headache and dizziness may precede increasing

shortness of breath, coughing, wheezing and pulmonary edema which may lead to death. In those who recover, the illness may take a biphasic course with a second stage of increasing pulmonary distress, associated with bronchiolitis obliterans, after a quiescent period of two to six weeks following the original episode (Tse and Bockman, 1970). In bronchiolitis obliterans, bronchi and bronchioles contain an inflammatory exudate which undergoes fibrinous organization with permanent narrowing of the airways in the most severe cases. Recovery occurs in the majority of cases, especially with corticosteroid treatment, but some individuals appear to develop persistent functional abnormalities (Horvath et al. 1978), resulting from fibrosis or bronchiolitis obliterans. The long-term sequelae of toxic NO_x inhalation relate to the extent of exposure and severity of bronchiolitis obliterans (Guidotti, 1978). The National Academy of Sciences (1977) noted that exposure to NO_2 concentrations of about 25 ppm is associated with bronchitis and bronchopneumonia with recovery; exposure to 50 ppm with bronchiolitis and focal pneumonitis with recovery; and exposure at or above 150 ppm with fatal pulmonary edema, bronchopneumonia or bronchiolitis obliterans.

Acute effects resulting from short-term chamber exposure in humans: Orehek et al. (1976) noted increased airway resistance as a result of exposure to NO_2 at 0.11 ppm for one hour in 3 of 20 asthmatics and in 13 of 20, there was increased sensitivity to carbachol (a bronchoconstrictor). Healthy male non-smokers exposed for 2 hours to 0.62 ppm had no consistent changes in pulmonary or cardiovascular function (Folinsbee et al. 1978). Individuals with chronic non-specific lung disease demonstrated decreases in arterial PO_2 and corresponding increases in alveolar to arterial PO_2 gradient at 4-5 ppm (15 minutes) but not at 2 ppm; also significant increases in airway resistance occurred at NO_2 concentrations down to 1.5 ppm. Increased airway resistance occurred in healthy humans at 2.5 ppm (von Nieding and Wagner, 1979). Histamine release may play a role in triggering the effects on lung function after inhalation of

NO_2 (von Nieding and Wagner, 1979).

Long-term inhalation studies: Long-term inhalation studies in various species at or above 10 ppm have shown emphysema-like changes in alveolar walls, hyperplasia of type II pneumocytes and replacement of type I by type II pneumocytes. Chronic exposure in rats to 2 ppm resulted in microscopic and ultrastructural changes including bronchiolar epithelial hypertrophy, loss of cilia and thickening of alveolar basement membranes (W.H.O., 1977). Guidotti (1978) summarized the animal studies by stating that prolonged exposure to NO_2 results in interstitial fibrosis and centrilobular emphysema.

Long-term epidemiologic data in humans are limited. Studies of children in high and low ambient NO_2 areas near Chattanooga, Tennessee and of children in homes with gas stoves compared to those in homes with electric stoves have suggested that there are small decreases in pulmonary function and increased rates of respiratory illness and symptoms in those with high NO_2 exposure. However, Parkes (1982) considered that there was no adequate proof to date that intermittent exposure to NO_2 causes emphysema in humans.

Combination with other pollutants (ozone): *In vitro* studies of human red cells (Goldstein, 1976) and *in vivo* studies of resistance of mice to streptococcal pneumonia (Ehrlich et al. 1977) suggest that the effects of combined exposure to ozone and NO_2 are additive, and possibly in some experiments, synergistic. However, controlled human studies have not suggested interactions or synergism (U.S. E.P.A., 1984).

Welding processes where encountered: oxides of nitrogen (NO_x) are formed during welding by the direct oxidation of atmospheric nitrogen at the high temperatures produced by

the arc or flame, as illustrated below (Villaume et al. 1979):

$$N_2 + O_2 \longrightarrow 2\ NO$$ (significant at temperatures greater than 1000°C)

$$2NO + O_2 \longrightarrow 2\ NO_2$$

NO_x exposures of concern can be encountered in greater concentrations in flame processes in general (cutting, gouging, oxygas welding) rather than in arc welding, particularly in confined spaces. Among arc welding processes, the highest mean exposures were found for GTAW on aluminum, followed by GMAW on aluminum and shielded metal arc welding (SMAW) on stainless steel (Ulfvarson, 1981). The same comments regarding reflectivity of base metal and use of shielding gas hold as for ozone. Approximate exposure concentrations found during welding are found in Appendix I.

1.3 Carbon Dioxide and Carbon Monoxide

These gases are formed by the decomposition of organic compounds in electrode coatings and cores, from inorganic carbonates in coatings and primers, and in flame cutting processes especially in confined spaces. CO is also formed by decomposition of CO_2 in the gas-shielded metal arc process. CO_2 is not considered to be a significant problem in most welding (Tinkler and Graville, 1983). The most serious CO hazards result from the reduction of CO_2 used in gas metal arc welding. Cutting and gouging operations, particularly those using combustible gases, can produce CO levels approaching threshold limit values well after total fume limit (recommended or promulgated standards for exposure to total welding fume) is reached (Tinkler and Graville, 1983).

1.4 Gases formed from Decomposition of Chlorinated Hydrocarbons

Chlorinated hydrocarbons can be present either in the workshop air or in residual amounts on surfaces following the degreasing

of metal parts. Irritating and toxic gases, notably phosgene but also dichloroacetyl chloride (DCAC) can be formed through photochemical oxidation by UV radiation of chlorinated solvents such as trichloroethylene, perchloroethylene and, to a lesser extent, methyl chloroform (1,1,1-trichloroethane) (Dahlberg and Myrin, 1971; Dahlberg, 1973; Andersson et al. 1975). These gases are severe deep respiratory irritants. Phosgene is associated with effects ranging from irritation of the throat, to dyspnea, and pulmonary edema, depending on duration and severity of exposure; DCAC with cough and eye irritation. The main hazard is that the irritant properties of phosgene do not provide sufficient warning of exposure but the penetrating odour of DCAC [plus chlorine or hydrogen chloride which may also be associated (Newhouse and Murray, 1981)] may cause the welder to take protective action (Dahlberg, 1971). Human exposures to phosgene reported in the literature consist of instances of acute overexposure. However, the work by Galdston et al. (1947) suggested that repeated exposures can result in residual pulmonary problems (emphysema).

Processes where encountered: The stronger UV radiation from GMA-type electrodes produces more rapid oxidation than that from non-consumable electrodes (Dahlberg, 1971).

1.5 Gases derived from coatings

Irritants such as acrolein and formaldehyde from alkyl and epoxy resin metal coatings and isocyanates, plus other decomposition products from polyurethane coatings, can also be evolved (Parkes, 1982).

It is important that the welder make every effort to learn the nature of the coatings on the base metals with which he is working and to take appropriate precautions if the coatings are unknown or may evolve potentially harmful decomposition products.

2.0 Particulates

The solid phase in welding fume can originate from a number of sources including the base metal, the electrode or filler material (and flux), and protective coatings such as metal base paints, galvanized surfaces or polytetrafluoroethylene. The major source of fume during SMAW (stick welding) is the metallic part of the electrode and its coverings, which in turn, depends on electrode type (acid, rutile, basic, neutral). The particulate consists largely of metal oxide particles from the base metals and filler materials. Fume generation rates depend on a large number of factors which have been reviewed elsewhere (by Tinkler and Graville, 1983 and by Hatch Associates, 1985 - Part 1, Section 4.0), but tend to be greatest with SMAW and FCAW (Palmer, 1983). Ulfvarson (1981) categorized welding processes by their geometric mean total dust contents into highest (GMAW on aluminum, SMAW on steel; about 10 $mg/^3$), intermediate (SMAW on stainless steel, GMAW on steel; about 5 mg/m^3) and lowest (GTAW; about 1-2 mg/m^3). High fume concentrations may also be generated in plasma and carbon arc cutting processes. Thus, there are wide variations in exposures depending on the welding technology used, base materials, job type, and individual factors, and welders may be exposed to average fume concentrations of about 2-10 mg/m^3 during 8-hour shifts (Stern, 1983a). However, welding fume concentrations up to 100 mg/m^3, particularly in confined spaces, have been reported (Stern, 1981a). It should be noted that the conditions in which welding takes place (open air, normal workplace, confined space) and the presence of specific toxic constituents may require, in certain circumstances, more attention to protection and prevention through engineering and education than just the control of total fume. Most of the particulate in welding fume tends to be respirable (Palmer, 1983).

Brief reviews of selected particulate constituents follow. There is the potential for interactions between the constituents, although this has not been well documented. These reviews are not intended to cover all possible minor

constituents; some have been the subject of previous health effects documents prepared by the Ministry of Labour.

2.1 Chromium

The effects of chromium have been reviewed in detail by a number of authorities. A criteria document on the effects of chromium on health has been prepared for the Ministry of Labour and should be referred to for detailed consideration of this area (Nieboer et al. 1984).

Chromium is a hard, brittle, lustrous steel-grey metal which is very resistant to corrosion and exists in a number of valency states including elemental chromium, trivalent chromium [Cr (III)], and hexavalent chromium [Cr (VI)].

Acute effects associated with occupational exposure to dust or mists containing hexavalent chromium include irritation of mucous membranes (upper and lower respiratory tract), resulting in rhinitis, irritation of throat, and if in higher concentration, cough, dyspnea, generalized bronchospasm and acute pneumonitis. Sensitization leading to asthma has been reported but occurs infrequently. Chronic effects on the respiratory system associated with hexavalent chromium include ulceration and perforation of the nasal septum, perhaps due to chronic exposures as low as 15-20 $\mu g/m^3$. Although pneumoconiosis and fibrosis have been infrequently suggested to occur in association with some chromium-related occupations, it is generally considered that the evidence for non-malignant chronic pulmonary disease from long-term exposures to chromium compounds is not strong and this aspect is open to question (Levy, 1985).

Skin effects that can be produced include both irritation and allergic contact dermatitis (through delayed hypersensitivity mechanisms). The primary irritant dermatitis (hexavalent chromium) has been associated with skin ulcers referred to as "chrome holes" or "chrome sores". Chromium is the second most common metal skin

allergen in the general population (nickel being most common) and chromates are the most common industrial sensitizers in men. Thus chromates are strong skin sensitizers. Although the skin hypersensitivity has, in the past, been unequivocally associated with Cr (VI), both hexavalent and trivalent forms of chromium are allergenic. The former is more potent as an inducer and elicitor of skin reactions.

Occupational exposure to chromium has been clearly shown to be associated with increased risk of lung cancer in the chromate manufacturing (hexavalent and trivalent exposures) and chromate pigment industries (sparingly soluble lead and zinc chromate exposure). There is also a suggestion of increased lung cancer risk in chrome platers who are exposed primarily to highly soluble chromates (chromic acid) and possibly in the ferrochromium industries where exposure is primarily to trivalent chromium and chromium metal (Nieboer et al. 1984) IARC (1982) considered that the evidence for carcinogenicity to humans was sufficient (i.e. that there is a causal relationship between the agent and human cancer) and considered chromium and certain chromium compounds to be a Group 1 carcinogen (indicating the compounds were considered carcinogenic to humans). Cancer excesses at other sites have been reported (including gastrointestinal tract, laryngeal, nasal) but not consistently. The evidence for carcinogenicity to animals was also considered sufficient by IARC, with positive results (Levy, 1985) for the intermediate and sparingly soluble chromates such as strontium, calcium and zinc. Nieboer et al. (1984) concluded that sparingly soluble (hexavalent) chromates have recognized carcinogenic potency while evidence is mounting for soluble (hexavalent) chromates. Exposure to trivalent chromium compounds when not associated with exposure to hexavalent compounds has not been proven to be associated with an increased risk of respiratory cancer.

The mutagenicity of chromium compounds has been reviewed by Venitt (1985) and by IARC (1982). For hexavalent chromium,

which caused DNA damage, point mutations and chromosomal anomalies in a wide range of assays, the evidence for genetic activity in short-term tests was considered proven. The evidence for genetic damage for chromium (III) was considered inadequate (IARC, 1982).

There is no human evidence that chromium compounds are associated with birth defects and adverse reproductive outcome. Animal experiments have shown chromium compounds to be embryotoxic and teratogenic (Hemminki and Lindbohm, 1985) and spermicidal (Nieboer et al. 1984).

Welding processes where encountered: Chromium-containing fumes are a potential hazard during the welding of stainless, low and medium alloyed steels, and steel coated with zinc chromate primer. Significant amounts of such fumes can be produced during the SMAW and GMAW processes, the former producing a much higher percentage of chromium (VI), often exceeding the ACGIH TLV for chromium (VI). Stern (1981a) has focused on the approximately 10% of workers engaged in stainless steel welding as possibly having a high risk for lung cancer ("hot spot"). Evaluation of carcinogenicity in welders is discussed in detail below (Carcinogenesis: Part II). Approximate exposure ranges for chromium are found in Appendix I.

2.2 Nickel*

Nickel is a silver-white, hard, malleable, magnetic element, capable of a high lustre and resistance to corrosion.

Dermatitis due to metallic nickel, nickel-plated objects and nickel alloys (with the exception of stainless steel) has been recognized and nickel sensitivity is the most common cause of allergic metal dermatitis, particularly in women.

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* A document on the effects of nickel on health is being prepared for the Ministry of Labour.

Except for industrial causes, nickel dermatitis in men is rare (Fisher, 1973). Nickel-associated occupational asthma has been reported (McConnel et al. 1973). A case of nickel asthma in Canada was shown to have high titres of IgE and IgG antibodies directed to nickel complexed with human serum albumen (Dolovich et al. 1984).

In nickel refineries and electroplating shops, rhinitis and sinusitis have occurred from inhalation of soluble nickel aerosols.

Nickel carbonyl, $Ni(CO)_4$, is the only organic nickel compound that has been recognized as a cause of systemic toxicity. A toxic, volatile liquid, and an intermediate in the Mond process for nickel refining, it can cause acute poisoning. As described by the National Academy of Sciences (1975), nickel carbonyl poisoning may involve initial symptoms of headache, vertigo, nausea, and vomiting, then a possible latent period followed by cough, hyperpnea, cyanosis, weakness, slight increases in temperature, white blood cells and pulse, and chest signs of pneumonitis. Severe delayed symptoms develop insidiously 12-36 hours after exposure. Death has usually been attributed to respiratory failure and pulmonary edema.

Epidemiologic studies conducted among nickel refinery workers in Wales, Canada, Norway and Russia have found excess incidences of cancer of the lung, nasal cavity and possibly the larynx. IARC (1982) considered the evidence for carcinogenicity in humans sufficient and placed nickel refining in Group 1. For nickel and certain nickel compounds, the evidence for carcinogenicity in humans was considered limited (IARC, 1982) which indicates that a causal interpretation is credible, but that alternative explanations such as chance, bias or confounding, could not adequately be excluded. These compounds were placed in Group 2A indicating they are probably carcinogenic to humans. As has been noted in numerous sources, the identity of the nickel compounds that may induce cancer in nickel

refinery workers has not been definitely established but attention has focused on insoluble aerosols of nickel subsulfide and nickel oxides (furnace dust), on the vapour of nickel carbonyl, and on aerosols of soluble nickel compounds (Sunderman, 1980; Levy, 1985). Animal studies have demonstrated that certain nickel compounds are carcinogenic in experimental animals including nickel subsulfide, and to a lesser extent, nickel carbonyl (Levy, 1985), and the evidence for carcinogenicity to animals for nickel and certain nickel compounds was considered sufficient by IARC (1982).

The evidence for mutagenicity (that is, for activity in short-term tests for genotoxicity) was considered in 1982 by IARC to be inadequate for nickel and certain nickel compounds. In a review considering more recent work, Venitt (1985) concluded that there seemed little doubt that nickel compounds are not totally devoid of biological activity which might be considered as "genotoxicity" but that compared to hexavalent chromium, divalent nickel compounds produce rather weak and inconsistent effects.

Experimental work has demonstrated that water-soluble compounds ($NiCl_2$, nickel acetate) have induced resorptions, malformations of several organs and decreased fetal weight in mice and hamsters at doses of 1-10 mg Ni/kg body weight (Hemminki and Lindbohm, 1985). Malformations were induced in rats and hamsters by nickel carbonyl. Nickel compounds can also interfere with male reproduction in animals (Hemminki and Lindbohm, 1985). No human studies relating occupational nickel exposure to reproductive outcome are available.

Welding processes where encountered: Nickel is encountered in processes similar to those for chromium (welding of stainless, low and medium alloyed steels), plus those involving nickel-clad steel, nickel-based alloys and nickel-based electrodes. Stainless steel fumes contain quantities of both nickel and chromium; thus nickel has also been of

concern because of its relation to carcinogenicity in other settings (Langard and Stern, 1983). Approximate exposure ranges are found in Appendix I.

2.3 Fluorides

Fluoride compounds in welding fume may be associated with upper respiratory tract irritation. Although repeated exposure to high levels of fluoride can produce systemic effects including bone disease (fluorosis or osteoscleroris due to deposition of fluoride), this is very rare. Crippling fluorosis from (any) industrial exposure has not been reported in the United States (Proctor and Hughes, 1978).

Welding processes where encountered: Welding fumes containing fluorides (usually as compounds of calcium or silicon) are generated during processes using basic (low hydrogen) electrodes, in particular with SMAW and FCAW (Tinkler and Graville, 1983). Although bone abnormalities in welders have not been documented, elevated urinary fluoride excretion and increases in urinary fluoride over the shift have been reported in welders (Smith, 1968). Correlation of the concentration of airborne fluoride with that of urine fluoride in exposed welders has also been reported (Pantucek, 1975). Recent work has documented a linear relationship between the airborne fluoride concentration and the post-shift urinary fluoride concentration in arc welders using basic (low hydrogen) electrodes (Sjogren et al. 1984).

2.4 Manganese

Acutely, manganese oxide can cause an influenza-like illness (metal fume fever); the usual form of chronic intoxication (manganism) is associated with neurological and psychological manifestations including similarities to Parkinsonism (Finkel, 1983; National Academy of Sciences, 1973). Human and animal studies attempting to link chronic

effects of manganese and welding have focused on the central nervous system and are discussed in Part II of this document (Non-respiratory effects-nervous systems effects).

Processes where encountered: Manganese oxide can be encountered as a flux agent in the coatings of SMA electrodes, in the core of FCA electrodes, and particularly as an alloying element in the steel in electrodes or in the steel being welded (Villaume et al. 1979). Manganese can be found in both mild and stainless steel welding fumes.

2.5 Zinc (and other metals)

Freshly formed oxides of a number of metals that can be encountered during welding including cadmium, cobalt, copper, iron, magnesium, manganese, mercury, nickel, and especially zinc, can cause metal fume fever (See the section on Metal Fume Fever in Part II: Acute Effects).

Welding processes where encountered: especially when welding or cutting galvanized or zinc primed steel.

2.6 Copper

In addition to being a possible cause of metal fume fever, copper fumes can be associated with irritation of the upper respiratory tract and complaints of a metallic taste. Transient irritation of the eyes has followed exposure to a fine dust of oxidation products of copper produced in an electrode arc (Grant, 1974).

Welding processes where encountered: Copper is found in non-ferrous alloys including brass and bronze, as coatings on filler wire, and sheaths on air carbon arc gouging electrodes. Tinkler and Graville (1983) noted that significant exposure can occur when welding with copper-based electrodes for the welding of copper alloys or during brazing operations. Approximate exposure ranges are found in Appendix I.

2.7 Beryllium

Exposure to beryllium and its compounds has its major effect on the respiratory system. The acute form (acute beryllium poisoning) was described following outbreaks in Ohio and Massachusettes in the 1940s with the manufacture of beryllium oxide from beryl ore and in the fluorescent lamp manufacturing industry. Acute poisoning presents as a chemical pneumonitis with progressive dyspnea and dry cough (Finkel, 1983; Eisenbud et al. 1948). Chronic beryllium disease (that persisting for a year or more) resembles sarcoidosis with symptoms of slowly progressive dyspnea, fatigue, unproductive cough, and characterized by remissions and exacerbations. This chronic disease may have an immunologic basis (Devdhar et al. 1973). The criteria for diagnosis were reviewed by Kazemi (1980) and the epidemiology of beryllium disease by Hardy (1980). In addition, there are suggestions that exposure to beryllium may be associated with an increased risk of lung cancer (Wagoner et al. 1980; Infante et al. 1980). The International Agency for Research on Cancer (1982) classified beryllium and certain of its compounds within category 2A (ie. with limited evidence for carcinogenicity to humans and sufficient evidence for carcinogenicity in animals).

Processes where encountered in welding: Beryllium is present in some copper and steel alloys (e.g. beryllium bronze). Aluminum rods may contain traces of beryllium. Although there is the potential for exposure, according to Tinkler and Graville (1983), this does not occur in common welding practice.

A case has been reported by Roussel and Pottier (1978) of increasing dyspnea on exertion and a diffuse miliary x-ray picture in a 36 year old welder with 19 years experience. He welded for some time using electrodes with coatings containing beryllium, including welding in a confined space for several months prior to illness. Lung biopsy and patch

test with beryllium sulfate led to a diagnosis of pulmonary berylliosis.

2.8 Iron/Iron Oxide

Iron, the main particulate constituent of welding fume from non-alloy steel, is considered during the general discussion of welding fume (see the section on chronic respiratory disease - siderosis, Part II).

2.9 Barium

Barium compounds can produce irritation of the eyes, mucous membranes and skin, while inhalation of (insoluble) barium compounds has been associated with a benign pneumoconiosis, baritosis (Proctor and Hughes, 1978). Soluble compounds of barium are muscle and nerve stimulants. Non-intentional systemic poisoning in humans has only very rarely been reported.

Processes where encountered: Barium is encountered as a flux constituent in cast iron electrodes and in flux-cored wires for ferrous welding. It is of interest because of a relatively low ACGIH Threshold Limit Value of 0.5 mg/m^3 for soluble compounds, based on the intent of controlling exposure to barium nitrate (Dare et al. 1984). Dare et al. (1984) have shown that fume from a core-wired electrode used with a barium fluoride flux contained more than 16% soluble barium; this was associated with elevated barium urinary levels in exposed welders. They showed that limiting total fume might not control barium exposures in such situations.

2.10 Other Metals (including those potentially extraneous to the welding process itself)

There is potential exposure to other metals and their oxides during welding including aluminum, lead, cadmium, and mercury. The health effects of the latter three have been summarized by health effects documents prepared by the

Ministry of Labour. As lead and cadmium can be present in metal coatings and paints, these contaminants are potentially "extraneous" to the welding process itself.

Exposure to lead can occur during welding of lead coated metals (often from anticorrosive paints containing lead oxide), primers containing lead (zinc primers may also contain lead as an impurity), electrodes with lead coatings and during cutting operations on leaded steel. Lead poisoning from welding is rare but has been described (see section on non-respiratory effects: Part II).

Exposure to cadmium occurs with fume generated from welding of cadmium coated, plated or painted materials, or by brazing with silver-cadmium-based filler material. Cadmium-plated metals can be mistaken in appearance for galvanized metals.

2.11 Silica and Asbestos

The health effects of these materials have been summarized in health effects documents prepared by the Ministry of Labour and will not be reviewed here.

Processes where encountered during welding: Stern (1981a) has stated that "neither asbestos nor crystalline silica appear in welding fumes". However, lists and figures (such as Figure 1) purporting to describe all possible welding contaminants usually include both of these compounds.

How does one resolve these seemingly contradictory statements? First, it is known that silicon as silica, ferrosilicate, kaolin, feldspar and mica can be present in electrode coatings or flux and that aluminum alloys and low alloy steels also contain alloying silicon. Thus silica in some form may be present in fume. Villaume et al. (1979) noted, however, that while crystalline silica was not detected in fume, amorphous silica or soluble silicates were. Parkes (1982) noted that welders may have been

exposed intermittently to free silica dust if they worked near fettling or sandblasting operations in foundries ("bystander exposure"). Any free silica present in fume would combine in the arc with oxides to form silicates (Doig and Duguid, 1951).

Asbestos had been utilized as a flux component in the past and may still be found in small amounts in some electrode coatings (Tinkler and Graville, 1983). These authors stated, however, that the welding arc tranforms the asbestos to a harmless amorphous slag. As with silica, welders may have been exposed to asbestos dust when working near insulation workers (bystander effect).

Summary: In the report by Hatch Associates (1985; Part 1, Section 8.2) it was noted that the following processes "present the greatest opportunity for potential hazards, based on estimated frequency of use in industry":

Process	Potential Exposure
SMAW	Excessive fume concentration and fluoride (mild steel); chromium and nickel (stainless steel).
GMAW	High fume concentrations; excess ozone with stainless steel and aluminium.
FCAW	Excessive fume levels.
Air carbon arc/ Plasma arc cutting	Excessive fume; noise.
Oxy-gas cutting	Production of carbon monoxide or oxides of nitrogen.
Thermal spraying	Excessive fume levels of sprayed metal, e.g. zinc.

Although there are other rare exceptions, it is apparent that welders may experience high process-dependent exposures in a limited number of settings. These include SMA/SS (and, to a lesser extent, GMA/SS) where exposures to hexavalent chromium may occur; and GMAW with aluminum and stainless steel where considerable ozone exposures may occur. High exposures to NO_2 can occur in confined spaces and exposures to a number of compounds can occasionally occur with the use of specific consumables or from surface coatings extraneous to the welding process.

It appears that in most cases, total fume becomes limiting before most contaminants become limiting (Stern 1981a), with, perhaps, the exceptions of the process-dependent exposures noted above. Data presented by Stern (1981a), and by Jenkins et al. (1981) in a report from the Welding Institute in the United Kingdom (where breathing zone samples were taken inside the helmet), showed that significant proportions of welders are at risk to exceed recommended levels (eg TLVs) for various substances, including total fume. However, it was emphasized by both groups of investigators that with few exceptions, component element TLVs were not exceeded if the TLV for total fume (5 mg/m^3) had not been exceeded. This was also noted by Hatch Associates (1985) in their summary (Part 2, Section 5.7) of walk-through and air monitoring surveys in Ontario.

Part II.

HEALTH EFFECTS OF WELDING FUME

1.0 RETENTION AND CLEARANCE OF WELDING FUME

1.1 Studies in Animals

Lam et al. (1979) examined the biological fate (deposition and clearance) of fume following its entry into the lungs of rats and guinea pigs. The amount of fume, mean particle size and composition of specific metals (chromium, cobalt, iron, nickel, zinc) were determined in guinea pigs exposed to a fume concentration of 990 mg/m^3 for 256 minutes (GMAW of stainless steel) and rats exposed to a fume concentration of 1,178 mg/m^3 for 46 minutes (SMAW, flux-cored electrodes). Compared to the amount present immediately after termination of exposure, there was a marked decrease with time in the amount of fume found in the lung after exposure ceased. In rats, for example, iron decreased from 2,185 µg/g lung tissue immediately after exposure, to about 50% (1,085 µg/g) at 30 hours, and 657 µg/g at 30 days. For cobalt, the figures were 0.28, 0.12, and 0.09 µg/g, respectively. In guinea pigs, the time to eliminate 50% of the iron content from the GMA stainless steel fume from the lung was much longer (about 40 days). Intratracheal injection of radioactive GMAW fume particles into guinea pigs revealed three phases of elimination involving mucociliary clearance (short biological half-life of about 0.6 - 0.8 days depending on the element); phagocytosis by macrophages (half-life about 5-8 days); and a slower third phase due to leaching of elements from particles into solution in tissue fluid (half-life 135 - 300 days).

Recent work by Hicks et al. (1983, 1984), although primarily designed to address the fibrogenic/toxic effects of arc-welding fume, also determined lung burdens of particulate in rat lungs at various times after single and repeated exposures to SMA/MS and GMA/SS fumes. The relative

concentrations of iron (the predominant constituent of deposits) derived from either type of fume did not differ significantly; there was initial rapid clearance over the first 7 days with concentrations falling to one-third to one-half of those initially found. Subsequent loss over the next 3 week period was much slower. Lung clearance was not completely effective and persistent residual deposits were found. Such residua resulting from single exposure inhalation were small and confined mainly to peribronchial accumulations in macrophage clusters. Deposits remaining after repeated inhalation were larger and more widespread, with nodular accumulations following intratracheal inhalation.

Chromium concentrations following single inhalation exposures were small and declined rapidly following exposure to mild steel welding fume but were ten times higher following exposure to stainless steel welding fume, although falling at about the same rate. Significantly elevated levels of silica (probably amorphous) were demonstrated after SMA/MS fumes but electron microprobe analysis detected little or no silica following SMA/SS fume.

In a series of experiments closely allied to studies in humans to be described below, Kalliomaki et al. (1983a,b,c) examined retention and clearance of stainless and mild steel in the lungs of rats. They examined rats exposed to GMAW-stainless steel (GMA/SS) and SMAW-stainless steel (SMA/SS) fume. The former fume has a chromium content of lower solubility, mostly in trivalent or metallic forms compared to the latter. Total iron did not undergo detectable clearance at all; there were high retention levels for both fume types. Exogenous iron cleared with a half-time of 40 days for SMAW and 50 days for GMAW fumes (Kalliomaki et al. 1983a). The accumulation of chromium in the lungs was very high and cleared slowly in GMA/SS, with a half-time of approximately 240 days versus 40 days with SMA/SS where more of the chromium was in a soluble form. Thus, the two fume types had different clearance patterns.

Kalliomaki et al. (1983b) also compared the lung retention and clearance of SMA/SS and SMA/MS fume in rats via inhalation. For the former, total iron concentration did not change with cumulative exposure while concentrations of exogenous iron, chromium and nickel correlated with cumulative duration of exposure. Lung clearance was similar for these elements with biological half-times for exogenous iron, chromium, manganese and nickel of 50, 40, 40 and 30 days, respectively. In rats exposed to SMA/MS fume, the total iron concentration increased with cumulative duration of exposure and did not decrease during follow-up. The lung clearance patterns for manganese and exogenous iron differed from that observed for stainless steel, incorporating fast components with half-times of 6 days for iron and 0.5 days for manganese, and slow components (35 days for exogenous iron). Measurement of the remanent magnetic field of the chest area, by magnetopneumography (a measure of the metal contaminants that have accumulated in the lungs), revealed that the alveolar retention of SMA/SS welding fume particles after four weeks was about 4 times higher than that of SMA/MS fume particles (Kalliomaki et al. 1983c). This confirmed in vivo measurements in welders (Kalliomaki et al. 1980, 1981a) in which dust burdens in SMA/SS welders were greater than those in SMA/MS welders (see following section). This increased retention was secondary to decreased clearance.

1.2 Studies in Humans

Magnetopneumography, as just described, is a non-invasive magnetic method for the measurement of ferromagnetic particles in the lungs. The subject's thorax is placed in an external magnetic field, the field is then removed and the remanent field produced by any magnetized particles within the thorax is measured.

Freedman et al. (1981), using this method, found that 11 shipyard steel arc welders had concentrations of ferrimagnetic mineral retention several orders of magnitude

greater than three machinists, 16 former asbestos insulators and 24 rural control subjects. These concentrations correlated with total years employed in welding ($p<0.01$) and with the radiographic evidence of small rounded opacities ($p<0.05$) but not with smoking histories of the subjects.

Kalliomaki et al. (1981a) also used magnetopneumography to estimate the amount of lung-retained metal contaminants in a number of occupational groups. They found that SMA/SS welders had the highest mean quantity (about 4 g dust per person), four times that of the second highest group, SMA/MS welders (about 1 g), while that of GTA welders, iron factory and foundry workers and stainless steel grinders was considerably lower (about 0.2 g). In all groups, there were high inter-individual variations in the amounts of retained contaminants, possibly due to differences in individual lung clearance. The slower clearance for stainless steel contaminants mirrored the experimental findings in animals described above.

Kalliomaki et al. (1981b) tried to correlate the levels of dust retained in the lungs (as estimated by the average remanent magnetic field) with end-of-shift urinary concentrations of chromium in 83 SMA and GTA stainless steel welders, divided into categories by the percentage time spent in SMA/SS welding. Urinary chromium was significantly correlated with percentage time spent welding SMA/SS ($p<0.001$) and with average remanent magnetic field of the chest ($p<0.001$). An index of cumulative exposure (exposure in years x percentage of time spent in SMA/SS) also correlated with average remanent magnetic field.

For mild steel contaminants, Kalliomaki et al. (1983d) summarized their studies on lung retention and clearance patterns among shipyard welders, estimated by magnetopneumography. The average clearance rate was 20% per year, corresponding to a half-time of 3.5 years (i.e. long-term residence of a portion of the deposits). As a constant level of lung contamination was achieved in about 5-10 years

of continuous exposure, a balance between retention and clearance was obtained during long-term exposure. Magneto-pneumography was considered a valuable tool for following retention and clearance of lung dust. However, it would not yet appear to be suitable for routine application.

1.3 Biological Monitoring

Biological monitoring or the evaluation of occupational exposure from an analysis of biological fluids, has been utilized to assess various elements in the welding environment. Fluoride and lead are considered elsewhere.

1.3.1 **Chromium**: A number of the studies that have assessed the relationships between chromium (total and hexavalent) in the air and in urine or blood have involved welders (Tola et al. 1977; Mutti et al. 1979; Kalliomaki et al. 1981b; Rahkonen et al. 1983; Sjogren et al. 1983). By following urinary chromium concentrations after exposure, it appears that there are two compartments (slow and fast) in which chromium is distributed. Tossavainen et al. (1980) using the data of Tola et al. (1977) on four stainless steel welders, estimated that the half-time for chromium in urine ranged from 15-41 hours while Welinder et al. (1983) estimated the median half-time for the fast component for chromium elimination in stainless steel welders to be 7 (range 4-35) hours. In SMA/SS welders, there are peaks in urinary chromium concentrations in response to daily exposure as well as increases (accumulation) through the work-week. Total elimination of chromium over a weekend or non-exposure periods did not occur suggesting an increased body burden (Mutti et al. 1979), that is, there is chronic accumulation and excretion. These studies have assessed the relationship between urinary chromium and airborne chromium. Both end-of-shift urinary concentration and increase over the shift showed very good correlation with ambient hexavalent chromium (and usually total chromium). Some of these investigators have suggested that the best correlation of ambient chromium was found with the increase

in urinary concentration over the shift because of the contribution of long-term excretion. Relationships relating urinary chromium (in µg/L or µg/g creatinine) to corresponding airborne chromium levels have been developed as a result of these studies as well as non-welding exposures and are considered in the report on the Effects of Chromium Compounds on Human Health prepared for the Ministry of Labour (Nieboer et al. 1984). They will not be reviewed here. Blood or erythrocyte chromium did not add substantially to the information obtained. GMA/SS welding, where chromium content of fume may be higher but where there is only a small water soluble component, was not associated with marked changes in chromium in urine and blood.

1.3.2 **Nickel**: The amount of nickel in urine or blood of welders has generally been low. The average urinary nickel concentrations in welders of SMA/SS (Kalliomaki et al. 1981b; Rahkonen et al. 1983) or at a shipyard (Grandjean et al. 1980) were about twice those of controls. Bernacki et al. (1978) also found urine nickel concentrations in 10 arc welders (6.3 µg/L) to be significantly greater than hospital controls but this difference was not statistically significant when corrected to urinary creatinine. In GTA/SS welders, urinary nickel concentrations were not elevated (Kalliomaki et al. 1981b). The blood nickel concentrations in SMA/SS welders were not elevated relative to controls (Rahkonen et al. 1983). These authors also found that nickel concentrations in air were highly correlated with those in post-shift urine.

1.3.3 **Other metals**: Recent data (Chandra et al. 1981; Aitio and Jarvisalo, 1985) suggest that welders can be shown to have blood and urine manganese concentrations that are somewhat higher than in controls, but considerable overlap of values between welders and controls, as well as intra-individual variation, are present. The authors suggest that this may be related to the dietary intake of manganese. Such indicators of manganese in blood and urine are useful only on a group basis in indicating the level of exposure

but not in indicating individual exposure (or risk to health).

No data are available on the effects of welding fume exposure on the iron balance of man.

Suzuki et al. (1981) studied 12 welders working with cadmium-containing silver solder for 3 to 23 years, one year after cessation of solder use, as well as 5 control subjects. They found welders' urinary concentrations of cadmium, copper and zinc were 26, 2.4 and 2.6 times that of controls, respectively. Mean urine cadmium concentrations was 39.2 g/L and exceeded 50 g/L in three subjects.

Recent studies (Sjogren et al. 1983; Mussi et al. 1984) have shown preliminary evidence that urinary aluminum concentrations in welders working on aluminum were definitely higher than in control groups, and that on a group basis at least, urinary aluminum can distinguish exposed and non-exposed subjects, even at environmental concentrations at or below a time-weighted average of 5 mg/m^3. End-of-shift values were 2-3 times pre-shift values. These differences should provide information on exposure on that day while levels on Friday were higher than on Monday, reflecting exposure over the preceding week. There appeared to be a (linear) relationship of urinary aluminum with years of exposure (Sjogren et al. 1983) and with environmental aluminum concentrations (Mussi et al. 1984). Plasma aluminum levels in the exposed workers did not differ significantly from controls.

Summary: It is evident that urinary (and blood) levels of chromium and nickel are increased in stainless steel welders as are urinary aluminum concentrations of aluminum welders. It appears that the kinetics of these metals are affected in part by the welding process. GMAW fumes are cleared slowly from the lung while SMAW fume components are cleared more rapidly (Aitio and Jarvisalo, 1985). More knowledge is required before these levels in body fluids can be related to health effects.

2.0 TOXICITY IN EXPERIMENTAL ANIMALS

The sections on the toxicity of welding fumes in animals and in humans will serve to bring the potential health effects described in Part I into the welding context.

Exposure to iron fume is the most common metal exposure in welding coming from the vaporization of the base metal and from electrode coatings.

2.1 **Acute Studies:** Studies on various species including rats, mice, guinea pigs and rabbits have been reported. Experiments in the 1940s with rats involving fume exposure of 1000-2000 mg/m^3 for several hours in conjunction with various concentrations of oxides of nitrogen demonstrated pulmonary edema and death from bronchopneumonia (Villaume et al. 1979). Intratracheal injection and inhalation of iron oxide into the lungs by rats resulted in radiopacities comparable to "human siderosis". Histologic studies did not reveal fibrosis of the lungs (Doig and McLaughlan 1948).

More recently, Hewitt and Hicks (1973) exposed rats to short intermittent exposures from welding fume from rutile electrodes (30 minutes or 4 hours; 1500 mg/m^3) and found only minor histological signs of respiratory irritation including brown discolouration of the lungs (identified as fume deposits), yellow discolouration of the stomach, and large numbers of alveolar macrophages densely packed with particulate. Significant increases in iron, cobalt, chromium and antimony were observed, the concentrations of these elements in the lungs declining progressively over 75 days. No growth impairment of the animals was seen.

Following intratracheal instillation of fume from SMA welding of stainless steel, and of mild steel with basic and rutile-coated electrodes, White et al. (1981) found increases in lung weight, and inflammatory changes including increases in proportions of white cells, in pulmonary

surfactant and in hydrolytic enzyme activities which suggested that SMAW particles were cytotoxic in decreasing order: stainless steel > basic > rutile. This was followed (White et al. 1982) by studies to determine the role of the water-soluble fraction of hexavalent chromium in the production of the inflammatory changes. Most of the toxicity of stainless steel welding particles one week after instillation in rats could be related to the content of soluble Cr (VI), producing changes similar to those of potassium dichromate. The insoluble welding particle fraction produced demonstrable but fewer lung alterations. However, as noted by Palmer (1983), the effects were the same as the general responses to many types of inflammatory stimuli including those to flux-coated mild steel electrode fume. Thus, it was not possible to conclude definitively that the inflammatory effects were related to the Cr (VI) content.

2.2 **Chronic Studies**: Studies from Eastern Europe (summarised by Hicks et al. 1983 and Villaume et al. 1979) have described evidence of pulmonary fibrosis in rats and guinea pigs exposed for many months to fume concentrations (usually from coated electrodes) in the range of 50-100 mg/m^3. Other lung findings included increased collagen content, emphysema, iron deposits (siderosis) and thickening of lung septae without fibrosis. No particular fibrogenic agent was identified.

In detailed work, involving single acute and repeated administration of SMA-mild steel and GMA-stainless steel welding fume to rats with examination of both the initial and long-term effects, Hicks et al. (1983, 1984) noted differences between the two forms of fume. The lung damage observed fell into two categories: the initial (acute, inflammatory) and later (cellular) effects associated with persistence of deposits of particles. The initial changes of pneumonitis and bronchiolitis could have been attributed to the effects of inhaled irritant gases (ozone, nitrogen oxides) or to soluble toxic components leached out of

deposited particles. GMA/SS particle deposits were more persistent and lesions of pneumonitis more severe with inhibition of phagocytosis or clearance and damage to epithelial cells being associated with possible toxic effects in macrophages. Both fume types caused alveolar epithelial thickening with proliferation of type-II pneumocytes. Taking alveolar thickening as an indicator (which was more persistent and extensive with GMA/SS than with SMA/MS), the authors suggested that some additional effects resulted from GMA/SS inhalation consistent with a yield of soluble toxic components. They emphasized, however, that this effect could result from contact with dusts that range from the virtually inert to highly cytotoxic or fibrogenic.

Long-term effects, between 80 and 300 days, involved formation of nodular aggregates of particle-laden macrophages with giant cells. Inhaled deposits of SMA/MS fume were cleared from alveolar spaces producing low grade fibrotic (collagenized) changes but little or no residual damage to adjacent structures. However, nodules containing GMA/SS fume were irregular and surrounded by collapsed, thickened epithelium with effects (atelectasis and consolidation) on surrounding alveolar tissue but with no collagen fibrosis. Consistent with this finding, determinations of lung hydroxyproline, an amino acid found in collagen, were significantly elevated in animals given SMA/MS intratracheally (but less than doubled indicating only low-grade collagenization) while there were no significant changes in those given GMA/SS. Five- and ten-fold increases in lung hydroxyproline have been reported with cystalline silica. It is noteworthy that the greater toxicity (to macrophages) associated with GMA/SS compared to SMA/MS fume was not associated with a fibrogenic mechanism.

In summary, the authors contended that welding fume lies quantitatively between innocuous materials such as iron oxide, titanium oxide and carbon on the one hand, and highly fibrogenic particles (quartz and asbestos), on the other.

The two types of fume provoked distinct pneumoconiotic effects, each of low grade intensity.

Stern and Pigott (1983) and Stern et al. (1983) considered *in vivo* studies to indicate that iron oxide does not in itself produce fibrotic reactions, and suggested a weak potential fibrotic potential for Cr (VI) and/or other putative fibrogens. Considering the gaseous fraction of welding fume, they noted that the toxicity and fibrogenic potential of NO_2 had been demonstrated in a number of species: interstitial fibrosis from prolonged low-level exposure and bronchiolitis obliterans characterizing the residual effect of acute severe exposures (Guidotti, 1978). However, despite suggestions by these authors implicating hexavalent chromium as a fibrogen in welding fume, the evidence from other chromium-exposed working populations suggesting that long-term exposure to chromium compounds is associated with chronic lung disease is not strong. Moreover, their conclusion appears to contrast with that of Hicks et al. (1983). To date, no studies have been able to identify a specific *in vivo* fibrogen in welding fume.

2.3 **Carcinogenicity of Welding Fume in Animals:** Although studies have shown that various welding fume components may be carcinogenic (see Part I and Section 4.6 on carcinogenicity in humans), there is as yet no experimental evidence that welding fumes are *in vivo* animal carcinogens when given by the inhalation route (Stern 1983b).

2.4 **In Vitro Studies Relating to Cytotoxicity and Fibrogenic Potential:** *In vitro* tests can examine the effects of chemicals on cell function or viability. Studies relating to mutagenicity are reviewed separately.

White et al. (1979, 1980) compared the cytotoxic effects of saline-suspended fume particles from three welding processes (SMAW using rutile-covered stainless steel electrodes; and basic or rutile mild steel electrodes), on different short-

term systems *in vitro*. Hexavalent chromium (nearly all soluble) represented about 3.5 per cent by weight of particles from the stainless steel process but only a trace from the other two. In the first cytotoxicity study, in which a rapidly-dividing human cell line was cultured in the presence of various concentrations of the welding fumes, the stainless steel welding fume particles were considered more toxic (causing decreased cell proliferation) than the other dust types. The dose-response relationship for this inhibition produced when hexavalent chromium was added to the culture was similar to that for the stainless steel dust, suggesting that the toxic effect of the fume was almost entirely due to hexavalent chromium. However, in another test, using rat alveolar macrophages, all the dusts produced similar 25-30% decrements in viability of the cells, suggesting that a component in the stainless steel particles, in addition to hexavalent chromium, appeared to be toxic to macrophages.

In a more recent report from these Norwegian investigators, White et al. (1983) exposed alveolar macrophages in culture to SMAW fume particles, chromium, chrysotile and titanium dioxide. Using trypan blue exclusion as a measure of cell viability, all the welding particles were cytoxic, the cytotoxicity increasing in the order: mild steel rutile-coated electrode < basic-coated electrode < stainless steel. Titanium dioxide was not found to be cytotoxic. Chromium (VI) in concentrations equivalent to that found in the stainless steel particles was toxic to macrophages but less so than the total particulate material while Cr (III) had no effect on viability. This toxicity was attributable to the Cr (VI) content as supported by the *in vivo* studies described above (White et al. 1981). The welding particles appeared to cause membrane damage without cell destruction.

Stern and Pigott (1983) used an *in vitro* fibrogenic activity assay (the rat peritoneal macrophage or RPM assay), to screen welding fumes to determine the presence of fibrogens. Four fume classes were tested: (1) mild steel;

(2) SMAW on stainless steel (SMA/SS); (3) GMAW on stainless steel (GMA/SS), and (4) nickel welding consumables. Positive (quartz) and negative controls were incorporated. The results showed the fibrogenic potential of classes 1 and 4 to be low, class 3 to be moderately high and only class 2 (SMA/SS) to show distinct fibrogenic potential. They felt that the potential source of fibrogenic activity was most likely hexavalent chromium, probably the insoluble fraction. It was noted that unlike SMA/SS fumes, in GMA/SS fumes, the chromium is mixed with iron as $Fe_xCr_yO_z$ which is relatively inactive (Stern et al. 1983). It should be re-emphasized, however, that the evidence suggesting that long-term occupational exposure to chromium compounds in other industries is associated with chronic lung disease is not strong.

3.0 ACUTE EFFECTS IN HUMANS

3.1 Acute Pneumonitis/Intoxications

Fatal and non-fatal pneumonitis have been frequently described in welders. Inhalation of ozone, oxides of nitrogen, phosgene and cadmium fume can cause acute pneumonitis, acute bronchitis and pulmonary edema. Acute eye and throat irritation from ozone and fluorides can be expected at exposures to concentrations lower than those producing pulmonary intoxications.

3.1.1 Ozone: Kleinfeld and Giel (1956) and Kleinfeld et al. (1957) described three cases of severe acute ozone toxicity (exposure about 9 ppm ozone) among 8 welders using argon-shielded gas metal arc welding with illness characterized by pulmonary edema. Recovery occurred in 1-2 weeks but with persistent complaints of exertional dyspnea in the two most severe cases. Other welders chronically exposed to 0.3 to 0.8 ppm complained of mucous membrane irritation, cough and headache. Levels of nickel carbonyl, phosgene and nitrogen oxides were said to be negligible.

Moles and Collins (1957) studied less severe effects of ozone exposure in GMA welders where workroom ozone levels exceeded 0.1 ppm in all determinations. Welders complained of eye and respiratory tract irritation when using a shielding gas mixture of 98% argon, 2% O_2 but not with pure argon, helium or CO_2. No evidence of residual effects was found after welding with the argon/O_2 mixture was discontinued. Challen et al. (1958) also described symptoms of upper respiratory tract irritation in 11 of 14 welders exposed to ozone at concentrations of 0.8 - 1.7 ppm. The symptoms no longer occurred when ozone concentrations were reduced to 0.2 ppm by improved ventilation.

In 1976, Kurta reported a case of acute pulmonary edema following GTA welding in an enclosed aluminum tank with inadequate ventilation and no respiratory protection. Recovery was rapid with return of lung function to normal within 11 days. No environmental measurements were reported.

3.1.2 **Oxides of Nitrogen:** Doig and Challen (1964), in a review of respiratory hazards in welding, noted that there were numerous reports in the literature of acute respiratory disease (pneumonia, lung edema) from exposure to nitrogen oxides formed by the use of oxyacetylene flames in enclosed spaces. Fatal cases due to electric arc-welding in enclosed spaces have also occurred. The exposures in some fatal cases have been very short (15-30 minutes). Little or no actual exposure data have been reported.

Norwood et al. (1966) described a case of pulmonary edema in a worker after several hours use of an acetylene torch for metal-cutting in a poorly ventilated space. Reenactment of the event produced NO_2 levels of 90 ppm in 40 minutes. Two of the four cases reported by Jones et al. (1973) involved pulmonary edema in workers engaged in oxyacetylene burning in a poorly ventilated tank. No exposure data were available.

Morley and Silk (1970) reviewed cases of "nitrous fume" gassing in the northern division of H.M. Factory Inspectorate in Britain, 1959-1968. Of the 11 cases, there were no welders but three were burning off metal in enclosed spaces. A survey of NO_2 concentrations at burning and welding sites, revealed all those collected during flame cutting to be above the ACGIH TLV (then 5 ppm). The potential hazard of "nitrous fume" during welding in confined or semi-confined spaces with insufficient ventilation was stressed.

Maddock (1970) estimated that NO_2 exposures exceeding 100 ppm preceded a fatality during oxyacetylene torch use.

3.1.3 Cadmium: Fatal cadmium-fume pneumonitis among welders has been reported by numerous authors including, for example, Christensen and Olson (1957) following welding of cadmium-coated metal; Blejer (1966) and Blejer et al. (1966) following brazing indoors with silver-cadmium alloy; Patwardhan and Finckh (1976) following several hours welding handles onto cadmium-plated drums; and more recently, by Lucas et al. (1980), following 30 minutes welding with an oxyacetylene torch and silver solder containing over 20% cadmium in a large building with open doors but no specific ventilation. The fatality among the five cases reported by Beton et al. (1966) engaged in cutting cadmium-plated bolts in a confined space with an oxyacetylene burner died from renal necrosis. Non-fatal cases of cadmium-associated pneumonitis in welders were described by Beton et al. (1966); Blejer et al. (1966) and Townshend (1968). The latter case involved the welding of "silver" plate (an alloy of 91% silver and 9% cadmium). At 17-year follow-up (Townshend, 1982), this man had dyspnea, crepitations, and restrictive ventilatory defect with reduced gas diffusion suggesting that progressive pulmonary fibrosis developed as a late result of the acute cadmium inhalation. Barnhart and Rosenstock (1984) recently documented the persistent restrictive ventilatory defect and long-term sequelae that can follow cadmium pneumonitis.

None of the previous articles reported environmental conditions except that by Townshend (1968) where the amount of cadmium in fume was determined to be 64 mg/m^3 and that by Blejer et al. (1966) where a study of a simulated outdoors (rather than indoors) work operation revealed atmospheric cadmium concentrations of 0.140 mg/m^3 in the breathing zone. It has been estimated that 2500 minutes-$milligrams/m^3$ is a lethal dose of cadmium oxide fume (Christensen and Olson, 1957).

3.1.4 Polytetrafluoroethylene (PTFE): Pulmonary edema has been reported (Evans, 1973) following the use of an oxy-acetylene torch to cut the cast iron pipe supports of a table which had been used to hold PTFE coated molds. The temperatures exceeded that required for decomposition; presumably PTFE pyrolysis products were involved.

3.2 Asthma

Occupational asthma has been described in welders but no estimate of prevalence or incidence can be made. Keskinen et al. (1980) reported five patients who developed asthmatic symptoms after SMA stainless steel (SMA/SS) welding and who demonstrated positive bronchial provocation tests on challenge with SMA/SS welding fume. Of three re-examined two years after cessation of exposure, one no longer reacted to welding provocation and two did. On re-challenge, these two patients developed reactions to SMA/SS fume but not to lactose, SMA/MS or GMA/SS fume. It was felt that the remarkably persistent asthmatic reactions in the absence of further exposure were caused by either chromium or nickel in the SMA/SS fume but specific challenges with these metals were not performed.

3.3 Metal Fume Fever

Metal fume fever (MFF) is a relatively common, acute febrile illness of short duration that occurs during welding operations. The condition results from the inhalation of minute particles of oxides of various metals. Freshly

formed fumes are thought to cause the disease although it is the size of particles that is the important factor in producing the illness (Pierce, 1983). Zinc, copper and magnesium are most commonly implicated but cadmium, iron, manganese, nickel, selenium, tin and antimony may also be responsible. The fume may be derived from metal coatings and galvanized surfaces, as well as from base metals and consumables. The symptoms, which may simulate a flu-like illness, come on 4 to 8 hours after exposure, and include thirst, dry cough and throat, metallic taste in the mouth, chills, dyspnea, malaise, muscle aches, headaches, nausea, and rarely vomiting. Fever and chills may be followed by sweating and possibly prostration but these resolve by the next morning and the worker can return to work. Signs include a body temperature rise to 38^0 to 39^0C (reaching a maximum 10-12 hours after inhalation), and polymorphonuclear leukocytosis (Parkes, 1982; Finkel, 1983; Morgan and Seaton, 1984). MFF may occur on the first day a new employee works.

Less is known about the mechanism(s) which produce the MFF syndrome. There have been suggestions that it is an "allergic" entity (Villaume et al. 1979; Newhouse and Murray, 1981). Before discussing the evidence for this, one should review the features suggestive of an allergic, idiosyncratic or hypersensitivity basis for disease in general, which include a low incidence; a latent or sensitization period; and perhaps, unpredictability or the inability to reproduce lesions in experimental animals.

McCord (1960) has been frequently quoted as labelling MFF as an immunological disease. However, McCord noted that since a workman can exhibit the syndrome on the first day of exposure, "allergic phenomena--may be ruled out", and that "histamine may be--caused to be mobilized in the body in the absence of antibodies". However, he postulated that allergen-antibody complexes (with metal oxide particles presumably acting as part of the allergens), and autoantibodies could result in an allergic response whenever exposure to the metallic oxide recurs. He admitted that "none of these postulates had been proved". The absence of

symptoms in welders for several days after an attack, despite continued exposure, is suggestive of a temporary "immunity". The time course, chills, fever and leukocytosis resemble hypersensitivity pneumonitis. Some metals such as nickel, chromium and beryllium found in welding fume are known to be associated with immunological reactions involving both the lung (Anonymous, 1984) including asthmatic reactions and the skin (allergic contact dermatitis).

However, in 1975, Fishburn stated that as yet, McCord's postulates that MFF is an immunological disease remained unconfirmed. Piscator (1976) also reported that the mechanisms were not fully understood but an allergic etiology was not mentioned. Johnson and Kilburn (1983) noted that no theories regarding the pathogeneses had been proved. Parkes (1982) noted that MFF is probably related to minute fume particles which reach the alveoli, possibly associated with the production of endogenous pyrogens.

Specific features of MFF, namely, that it can be reproduced in experimental animals (Morgan and Seaton, 1984; Pierce, 1983), that it can be experienced on the first day by a new employee (no latent period required) and that large proportions of the welding workforce (about 30% are affected; Ross, 1974) are not consistent with features suggestive of an allergic basis for disease.

Thus, in summary, while MFF has manifestations resembling an immunologic disorder, definitive evidence is lacking both for the exact mechanism, and if the mechanism does prove to be on an immune basis, for the specific allergens involved. Conceivably, the mediators leading to the manifestations of chills, fever and leukocytosis, could be released by a direct (toxic) pharmacologic interaction of human cells with some component in the welding fume and not necessarily by an allergic reaction. It is possible that the more recently recognized elements of the immune system, such as interleukins (e.g. endogenous pyrogen or Interleukin I) are involved but evidence is lacking at this time. Also

unanswered are why the syndrome develops in only some exposed individuals and why it occurs only on the first day back at work, but does not recur with continued exposure.

Environmental data relating zinc oxide fume to MFF are sparse. Although MFF occurs below the threshold of 15 mg/m^3 proposed by Drinker et al. (1927), NIOSH (1975) noted that suggestions of MFF occurring in the range of 5 mg/m^3 were unsupported by firm data. ACGIH (1980) included personal communication of reports of MFF associated with concentrations below 5 mg/m^3, and retained the recommendation based on its TLV of 5 mg/m^3 established in 1962, considering that the incidence of attacks would be low.

MFF has many similarities to polymer fume fever associated with exposure to the heat-degraded polymer, polytetrafluoroethylene (PTFE), the causal mechanism of which is also not known.

3.4 Acute Changes in Pulmonary Function

In humans, acute exposure to welding fume under workplace conditions has been studied by examing pulmonary function changes before and after exposure but only small changes have been detected.

McMillan and Heath (1979) examined such acute changes in 25 SMA welders and controls selected from a population of electrical fitters working in a welding-free environment who were individually matched by race, sex, age and smoking habit. The doctors and laboratory staff were kept unaware of the man's occupation until after lung function tests had been performed. There were no significant differences in acute changes in FEV_1 (forced vital capacity), MEF_{50} and MEF_{25} (maximum expiratory flow rate at 50% and 25% respectively, of vital capacity) between the group of welders and controls, or of smokers and non-smokers. However, among the welders, there was a positive correlation ($p<0.05$) between the change in residual volume and total

fume, respirable fume, iron oxide fume and NO_x (correlation coefficient ranged from 0.422 to 0.528).

Palmer (1983) made reference to a study of Japanese welders by Ohmori et al. (1980) in which exposure to ozone was found to have a definite effect on pulmonary function. Flow volume curves were measured for 68 healthy welders, 20 to 29 years of age, exposed to relatively high levels of ozone. Dust exposures and smoking habits were considered. There was no correlation between peak flow rate or maximum expiratory flow at 75% of vital capacity and ozone concentration. However, the maximum expiratory flow at 25% of vital capacity of welders exposed to ozone concentrations of 1.05 ppm or more was significantly lower than that of welders exposed to less than 1.05 ppm. This is not surprising since this represents a level ten times the TLV.

Hayden et al. (1981) found no significant change in carboxyhemaglobin, spirometric measurements. mid-expiratory flow, density dependence of flow or closing volume measured before and after 15 minutes continuous electric arc welding. Exact exposures were not reported but continuous monitoring showed NO_x, ozone and CO were "low" relative to threshold limit values. There appeared to be little risk of acute respiratory disease when welding under the conditions of the study.

3.5 Pneumonia Morbidity and Mortality

Pneumonia represents an acute to subacute infectious disease process. Welders have been shown in a number of reports to have an excess burden of illness associated with pneumonia.

One source of data is the Decennial Reports from the United Kingdom. Doig and Challen (1951) reviewed the Decennial Report for 1951 and found that among welders there was a slight excess above that expected for deaths due to all causes (1196 vs 1092, SMR 110) of which a substantial part was due to pneumonia (70 observed, 31 expected; SMR 226). From the 1961 Decennial Report, the SMR for pneumonia for

welders, cutters and brazers remained elevated at 184 (101 observed, 55 expected) with an all-cause SMR of 107. Challen (1974) showed that this increase was not observed in the wives of welders, as seen below:

Standardized Mortality Ratios for Welders and their Wives in England and Wales Aged 15-64

Cause of Death	Welders, Cutters and Brazers			Wives		
	No. of deaths Registered	Expected	SMR	No. of deaths Registered	Expected	SMR
All causes	2163	2016	107	836	816	102
Pneumonia	101	55	185	12	19	63
Bronchitis	138	120	115	15	18	83
Influenza	21	15	140	5	6	83

In the Registrar General's decennial supplement on occupational mortality, 1970 - 1972 (1978), the SMR for pneumonia in this group remained high (157) with an all-cause SMR of 122.

Beaumont and Weiss (1980) examined the mortality experience of 8679 members of a metal trades union including 3247 welders, from 1950 to 1976, by the historical prospective method. Despite a low overall mortality risk, the welders had a 67 per cent excess of pneumonia deaths (19 observed, 11.4 expected: $p<0.05$). They suggested that both nitrogen dioxide and ozone exposure may have been associated with the excess. The effects of gases in the welding environment (ozone and nitrogen dioxide) in increasing susceptibility to lung infection were reviewed in Part I (gases).

Considering morbidity studies, Antti-Poika et al. (1977) found no difference in the prevalence of reported pneumonia among 156 welders and 106 controls. Ross (1978) interviewed 926 welders with heavy engineering and shipbuilding backgrounds and 755 controls concerning past history of respiratory illnesses. No significant differences in the amount of pneumonia and bronchitis were detected between the

non-smokers of the two groups, or between smokers other than in the 50-59 year age group. In this group, welders reported significantly greater amounts of pneumonia and bronchitis.

4.0 CHRONIC EFFECTS IN HUMANS

4.1 Absenteeism

There are a number of variables other than those related to occupation that influence absenteeism (and in particular sickness absence). Thus, absenteeism is only an indirect measure of morbidity. With these limitations in mind, there is some evidence that welders have more sickness absence than comparison groups.

The problem has been addressed as part of a series of studies involving the health of welders in British naval dockyards (McMillan, 1979; McMillan and Molyneux, 1981; McMillan, 1981). The study compared absence patterns among 533 welders with two control groups: Group 1, 717 boiler-makers and 835 shipwrights (who did not weld but worked in areas where welding took place intermittently), and Group 2, 99 electrical fitters, 403 painters and 765 joiners (who were unlikely to have any exposure to welding). All men worked in Her Majesty's Dockyards for at least 6 months during a five-year period. Indices of frequency of absence (rate of new spells and annual inception rate of spells) and of severity (mean annual duration and length of spells) were calculated. Age-standardized sickness absence patterns were presented both unstratified (McMillan and Molyneux, 1981) and stratified (McMillan, 1981) for smoking status.

Absence attributed to all disease did not differ among the groups. For absence attributed to upper respiratory disease, welders had the greatest mean length of spells and annual duration of absence. For absence attributed to lower respiratory tract disease, welders, after age-adjustment, were away more often and for longer spells than controls.

The excesses in upper and lower respiratory tract disease were seen mainly in current smokers with little difference among non-smokers

McMillan (1981) attributed these differences to welders' smoking rather than to exposure to welding fumes, stating that it was unlikely that these excesses indicated separate or clinically more severe illness attributable to exposure. Also McMillan claimed affected men became absent or maintained absence at an earlier stage in their symptoms than controls, possibly because they were more susceptible to the irritating and aggravating effects of welding pollutants during acute respiratory disease. He concluded that there was insufficient evidence to suggest that welding pollutants were the cause of the symptoms.

However, while this was a well-planned study, it must be recalled that these comparisons were between comparable smoking groups. The conclusions as to the role of smoking drawn from the study do not appear warranted based on the data provided. The data do suggest increased absence attributable to respiratory (especially lower) tract disease in welders who smoke.

Fawer et al. (1982) also examined sickness rates in a retrospective morbidity study of all welders (mainly electric arc) who had worked in a petrochemical plant for more than one year between 1970 and 1979. Adequate information was available for 36 of 37 welders who met the duration requirements. They were individually matched to controls at the same plant, of the same social class (pipefitters, boilermakers, mechanical and electrical fitters) on age, duration of employment, starting date, fitness at pre-employment examination and smoking habits. The welders had been exposed to welding fume for an average of 26 (range 4-42) years. Only certified absence (more than three days) was considered and similar indices of severity and frequency as described above were calculated. Absence indices for all diseases were similar in welders and

controls, but absences attributed to all respiratory diseases were slightly higher ($p<0.1$) in welders in severity, duration and frequency. The proportions of days lost (0.56 vs 0.24) and of number of spells (0.65 vs 0.40) for all respiratory diseases relative to that for all diseases were significantly greater in welders than controls. While the indices for upper respiratory tract diseases were similar in the two groups (Table below), for lower respiratory tract disease, welders had an average annual duration of absence 4.6 times greater, inception rate of spells 5 times greater (both $p<0.01$) and inception rate per worker 2.8 times greater ($p<0.025$) than the controls.

Absences Attributed to Upper and Lower Respiratory Tract Diseases

Absence Measurement	Upper Respiratory Tract Diseases		Lower Respiratory Tract Diseases	
	Welders	Controls	Welders	Controls
Average annual duration (days)	2.3	1.2	3.7	0.8 ****
Average length of spells	12.2	8.5	18.3	17.7
Inception rate of spells	0.19	0.15	0.20	0.04****
Inception rate per worker	0.50	0.47	0.47	0.19***

*** $p<0.025$

**** $p<0.01$

From Fawer et al. (1982)

Unlike McMillan (1981), these differences were seen in both smokers and non-smokers, although they were greater in the former. Finally to cover the possibility that welders take more sickness absences because they think of themselves "at risk", the authors examined absence attributed to digestive diseases (thought to be unrelated to welding fumes) and found no differences between welders and controls. Fawer et al. (1982) concluded that the results suggested that compared to controls, welders have more absences attributed to respiratory (especially lower respiratory) tract diseases, and that this is enhanced by smoking.

4.2 Respiratory Effects: Symptoms and Pulmonary Function

The limitations of studies discussed in the introduction apply especially to a consideration of possible effects of exposure to welding fumes on respiratory symptoms and on laboratory measures of pulmonary function. These limitations include the drawbacks of cross-sectional studies (including survivor effects of self-selection which can lead to an underestimation of health effects), inadequate control for confounding factors of smoking and/or other occupational exposures such as asbestos, and insensitivity of conventional pulmonary function tests (PFTs) which detect changes only in large airways.

Despite these limitations, there appear to be indications, initially from case reports (Charr, 1955) and confirmed in the epidemiologic studies to be reviewed, that exposure to welding fumes and gases may result in an excess of symptoms associated with chronic respiratory disease.

The criteria used in assessing the research on respiratory diseases associated with welding are given in Appendix II. The study groups are examined beginning in the next paragraph. The findings are then considered separately for symptoms (Section 4.2.2) and for Pulmonary functions tests (Section 4.2.3). Two such studies (Schuler, 1962 and

Morgan et al. 1963) that considered siderotics only and thus did not have subjects representative of all welders, and that did not control for smoking in any way, are not further considered. The study by Barhad et al. (1975) in which subjects and referrents with chronic pulmonary disease were excluded, was also not considered. All are cross-sectional in design. As noted in the Appendix, for many studies it was not possible to assess the adequacy of the questionnaires, or whether data collectors were blind to exposure status.

4.2.1 The Studies

Hunnicutt et al. (1964) studied 100 U.S. shipyard welders selected at random from employees who had worked 10 years or longer and were less than 60 years old. Mean duration of exposure was 18.8 years but exposures were not further characterized. A comparison group of 100 non-welders employed at the same shipbuilding plant were also selected at random. Confounding due to smoking was controlled and was more prevalent in welders than non-welders. However, smokers were defined as those giving a history of smoking one pack or more of cigarettes daily. Thus, it is possible the non-smokers included low consumption smokers.

Of 52 randomly selected welders at a metallurgical plant, Stanescu et al. (1967) in Rumania, selected 16 with radiographs suggestive of siderosis and over 7 years' exposure as arc welders. The mean duration of exposure for the men was 17 years, 10 of the 16 were non-smokers but no further details of exposure were given. The comparison group consisted of 13 individuals with no dust exposure. Symptoms were not reported for controls, and PFTs were not given for smokers and non-smokers separately.

Kleinfeld et al. (1969) studied 25 welders and 20 controls selected in an unspecified manner from a U.S. sheetmetal products plant using both stainless steel and "black iron ore". Mean duration of exposure was 19 years. Iron oxide

concentrations ranged from 0.65 to 1.7 mg/m^3 inside, and from 1.6 to 12.0 mg/m^3 outside the face shields, while previous sampling (1960-61) revealed iron oxide breathing zone concentrations ranging from 30-47 mg/m^3. The controls had no occupational dust exposure and similar proportions smoked. The analysis did not give separate results for smoking groups.

Fogh et al. (1969) studied 156 welders who had welded (mainly mild steel) for more than 5 years at Danish shipyards and plants manufacturing engines, boilers or tanks selected in collaboration with an inspector of factories. The 152 controls who were employed in sections of the plant where welding was not normally done, were initially selected by the plants but later this was changed to random selection. Symptoms for separate smoking groups were not presented.

Peters et al. (1973) studied 61 of 63 welders at a U.S. shipyard and 61 pipecoverers matched to the welders by age and duration of employment in the same shipyard. Height and smoking habits were not significantly different in the two groups. Mean iron oxide exposure for the welders was 4.3 mg/m^3 (range 0.3 - 18.6). As it was apparent that both groups shared asbestos and welding exposure, Boston policemen and pipefitters from another shipyard with minimal or no exposure to welding fumes or asbestos were also considered. The final analyses were not broken down by smoking habit.

Ulrich et al. (1974) studied 80 SMA shipyard welders and 80 age-matched controls selected from the shipyard who were not exposed to welding fumes. Fume exposures in semiconfined spaces ranged up to 340 mg/m^3 and in big halls up to 192 mg/m^3 but no mean exposures were reported. Average duration of exposure was 13 years.

Antti-Poika et al. (1977) compared pulmonary function and respiratory symptoms in 157 Finnish electric arc welders who

had been welding three years or more for at least three hours per day, and 108 referent workers "unexposed to welding fumes" who were comparable in age, smoking habits and social class. The welders mainly used covered electrodes and none had worked in shipyards. Thirty-nine per cent of welders had worked 15 or more years. The distribution of sampling results for total fume for a subset of the welders, given by the authors, ranged from 1-2.5 mg/m^3 for 20 welders, 3-4 mg/m^3 for 38 and 6-9 mg/m^3 for 30. These samples were obtained in the breathing zone but outside the helmet.

Ross (1978) reported on findings in 926 SMA welders with heavy equipment and shipbuilding backgrounds compared with a control group of all available craftsmen from other trades. A weakness of this study was that the data for the actual prevalences of respiratory symptoms and for pulmonary function were not reported.

Oxhoj et al. (1979) studied symptoms, spirometry, forced expiratory flows and closing volume in 119 arc welders and 90 controls from a Swedish shipyard. All welders had at least 5 (mean 17) years welding experience and controls were non-welding clerks (possibly of different socioeconomic status) employed at the same shipyard matched to welders on age, height and smoking habits. Selection was random for half the controls and non-random for the remainder. Mean total dust exposure exceeded 10 mg/m^3 in about half of the 60 personal samples obtained.

McMillan and Heath (1979) determined respiratory symptoms and baseline pulmonary function in 25 welders and control electrical fitters individually matched on race, age, sex and smoking habits as part of the study of acute changes described above. The controls were otherwise selected from a larger group by unspecified methods. The welders, 23 of whom had used SMAW almost exclusively, had a mean of 26 (range 6-43) years welding experience. This was the only study in which those performing interviews and tests were

blinded to the subjects' exposure status.

Akbarkhanzadeh (1980) studied all 209 welders who had been employed full-time in arc welding for at least one year (mean 22 years) in an United Kingdom shipyard. The controls consisted of 109 non-welders selected by unspecified methods exposed to the same general environment for at least 10 years but who had never been welders. Thus the selection criteria specified different duration of employment criteria for the exposed and referent groups. No information was provided on the environmental exposure. A weakness of the study was the inclusion of ex- and never smokers together as non-smokers.

Keimig et al. (1983) examined pulmonary function and symptoms in 91 mild steel welders at a U.S. heavy construction equipment manufacturer, aged 25-49, employed at the plant for at least 4 years (mean 110 months) preceding the study. Twenty-two persons welded outside the plant in apprenticeship programs at small manufacturing companies. The controls were hourly paid workers in assembly, machining and shipping at the small plant with minimal exposure to respiratory irritants and who had never been welders. The selected welders and controls were working on the first shift at the time of the study and were either lifetime non-smokers or current smokers.

Hayden et al. (1984) studied welders and controls engaged in maintenance or tool room work at three British factories making products for automotive industries. Controls were individually matched to welders on age and those who had previously been welders were excluded. Selection was by volunteering with 258 (71%) of 361 welders and 258 (38%) of 685 controls approached, participating. Thus this study is characterized by both poor response and a markedly different degree of response between groups. The duration of employment of each group was not specified.

McMillan and Pethybridge (1984) conducted what was described

as a case-control study but apppears to be cross-sectional with matched referents. The initial group consisted of 328 (89%) of 368 welders employed at three U.K. naval shipyards who responded to an invitation to participate (volunteer). The actual study group was 135 welders representing a random sample of the initial group, 45 years and over. Controls were skilled never-welding craftsmen employed in the dockyards and matched one-to-one for sex, smoking habit, age and potential exposure to asbestos.

Findings of the studies:

4.2.2 Respiratory Symptoms

Relative risks (estimated by prevalence ratios) and levels of significance for welders relative to referents have been calculated (Table 2).

Hunnicutt et al. (1964) found that in both smokers and non-smokers, the prevalence of all respiratory symptoms considered (cough, sputum, wheezing, dyspnea) exceeded that in non-welders, most by at least two-fold, but only the difference for wheeze in smokers was significant at the 5% level.

Reporting combined smoking categories only, no differences between welders and referents were found in prevalence rates by Kleinfeld et al. (1969) for chronic cough, dyspnea or wheezing, by Fogh et al. (1969) for chronic bronchitis, and by Peters et al. (1973) for chronic cough or shortness of breath.

Overall, Ulrich et al. (1974) found no differences in the prevalence of cough, sputum or nasal catarrh but dyspnea was more frequent in welders than controls (31% vs 15%, $p<0.05$). Among non-smokers, the prevalence rates of cough (44% vs 12% $p<0.01$), sputum (41% vs 12%, $p<0.05$) and dyspnea (19% vs 4%, $p>0.05$) were three to four times greater in welders than controls (Table 2).

Antti-Poika et al. (1977) found no significant differences between welders and controls in the prevalence of persistent cough (9% vs 10%) or dyspnea (29% vs 33%) but did find significant differences in simple chronic bronchitis (defined from modified Medical Research Council or MRC questionnaire as production of phlegm first thing in the morning and during the day for three months each year) (24% vs 14%, $p<0.01$). Stepwise regression was reported to show no relationship with smoking or level or time of exposure.

Ross (1978) reported no differences in symptoms in non-smokers or for smokers other than the 50-59 year age group where welders had significantly greater amounts of cough with sputum. This study did not report any actual age-specific prevalence rates but only the statistical results of comparisons.

Oxhoj et al. (1979) found increased symptom prevalence rates in welders compared to non-welders in both smokers and non/ex-smokers (Table 2). In the latter group, differences for cough and sputum (23% vs 6%, $p<0.01$), wheezing (19% vs 4%, $p<0.05$) and dyspnea uphill (37% vs 9%, $p<0.01$), were statistically significant.

McMillan (1979) found a similar prevalence of dypsnea but welders reported a non-significant increase in chronic bronchitis using the MRC questionnaire (5 of 25 vs 2 of 25).

Akbarkhanzadeh (1980) found increased prevalence rates for both smoking and non-smoking welders (Table 2). The prevalence of chronic bronchitis (18% vs 0%) in smokers and persistent cough and sputum in non-smokers (12% vs 2%) was significantly greater in welders than in the controls.

Among non-smokers, Keimig et al. (1983) reported a greater prevalence of respiratory symptoms in welders than controls as determined by self-administration of the American Thoracic Society questionnaire. Differences in the reported prevalence of phlegm (22% vs 6%) and episodes of cough and phlegm (13% vs 0%) were significant at the 5% level. There

were no significant differences in symptom prevalence between smoking welders and controls.

Among non-smokers, Hayden et al. (1984) found no differences in phlegm production between welders and non-welders. Among smokers, welders had a greater frequency of chronic phlegm production than controls ($p<0.05$) but as no prevalences were given, the rate ratio cannot be determined. There was no difference in the reported prevalence of cough between the groups.

McMillan and Pethybridge (1984) noted no significant differences between welders and controls with respect to the proportions reporting chronic cough, chronic phlegm or chronic cough and phlegm. However, dyspnea was more common in welders than controls (28% vs 7%, $p<0.05$) by unmatched analysis). A matched analysis was not possible from the data provided. Dyspnea was significantly more frequent in all smoking classes including non-smokers (5 of 23 vs 0 of 23, $p<0.05$ by Fisher's exact test).

Summary: The studies reviewed are consistent in suggesting an association of an increased prevalence of one or more chronic respiratory symptoms (dyspnea, cough, sputum, wheezing, persistent cough and sputum, chronic bronchitis) in welders compared to the available controls or to the general working population (Newhouse and Murray, 1981). Nine (69%) of the 13 studies revealed such evidence that was "statistically significant". Of these 9, seven reported data separately for non-smokers. Of these, four (Keimig et al. 1983, McMillan and Pethybridge 1984, Ulrich et al. 1974, Oxhoj et al. 1979) showed significant excesses in non-smokers for one or more symptoms. These associations were moderately strong with prevalence (rate) ratios generally about two to four. None of the studies effectively assessed or reported the rates by duration of exposure or derived cumulative exposure indices. As with cross-sectional studies generally, one cannot be certain that the exposure preceded the symptoms. Excess prevalence rates were found

in both shipbuilding and non-shipbuilding settings. It should be noted that among welders, smokers had symptom prevalence rates several fold greater than non-smokers. These symptoms may be compatible with "industrial bronchitis" (Morgan, 1978) or perhaps more correctly, chronic "simple bronchitis" (Parkes, 1982), associated with dusty trades in general, rather than being specific to welding. However this does not detract from the health effect itself.

There is not sufficient data to relate this finding to dose or duration of exposure or to a specific constituent within welding fume. It is noteworthy that in a cross-sectional study of stainless steel and mild steel welders (Kalliomaki et al. 1982), both groups had a similar frquency of respiratory symptoms.

4.2.3 Pulmonary Function

The findings on analysis of pulmonary function in these cross-sectional studies are not quite as consistent as those for respiratory symptoms. A summary is presented in Table 3.

Hunnicutt et al. (1964) reported that mean FEV_1 (forced expiratory volume in one second), PEFR (maximal or peak expiratory flow rate) and maximum mid-expiratory flow rate (MMEF or FEF_{25-75}) were significantly lower ($p<0.01$) in welders compared to controls. These differences were seen in smokers but not in non-smokers. Values were not corrected for height although the groups were similar in age and body surface area. Evidence of obstructive abnormality on spirometry (FEV_1 <90% predicted and FEV_1/FVC <70%) were found in 37% of welders and 10% of controls (41% vs 10% among smokers, 28% vs 10% among non-smokers). Thus, it appeared that both welding and smoking increased the likelihood of abnormality. There was no correlation between the degree of radiographic abnormality (siderosis) and impairment of pulmonary function. Kilburn et al. (1985)

noted that the fact that vital capacity was about one litre lower than predicted for both welders and controls, may reflect-asbestos exposure in both groups.

Stanescu et al. (1967) found no difference in FVC (forced vital capacity), FEV_1 or FEV_1/FVC ratio between welders (all with siderosis) and controls. Separate analysis by smoking groups was not provided. Welders had statistically significant reductions in static and dynamic compliance ($0.001<p<0.01$). Seven welders had compliance values below the lower limit of the 13 controls. This is a sign of decreased pulmonary distensibility, conceivably associated with fibrosis.

No differences between welders and controls were found by Kleinfeld et al. (1969) (in spirometry, lung volumes or diffusing capacity), by Fogh et al. (1969) (in FEV_1% of predicted), by Antti-Poika et al. (1977) (in FVC, FEV_1, or FEV_1/FVC) or Ross (1978) (in FVC, FEV_1, or PEFR). All of these reports except that by Ross (1978) did not analyze the data by separate smoking groups. Among welders, Kleinfeld et al. (1969) found no differences between those with more than, and those with less than 20 years experience. Antti-Poika et al. (1977) expressed the data in litres with no correction for age and height although the distributions of these variables were said to be similar. These study weaknesses should be borne in mind.

Peters et al. (1973) found no significant differences in spirometry, single breath diffusing capacity and thoracic resistance between welders and pipefitters. However, both the welders and these controls had lower FVC, FEV_1, and PEFR ($p<0.005$, 0.005 and 0.05, respectively) when compared to pipefitters engaged in new ship construction who had minimal or no exposure to asbestos and welding fume. These values for both groups were also low when compared to Boston policemen. They suggested that the data concerning welding effects were inconclusive.

Oxhoj et al. (1979) reported no differences between welders and controls in VC, FEV_1, MMEF, MEF_{50} and MEF_{25} (maximum expiratory flow at 50% and 25% of vital capacity). In non- and ex-smokers, closing volume (CV) and closing capacity (CC) were significantly higher and total lung capacity was significantly lower in welders compared with controls. Between welders and controls who smoked, there were no differences in these variables. CV and CC may be considered indicators of possible abnormal early changes in small airways. However, it is possible that the clerks who served as controls differed in other ways from the welders, but the influence of such differences on the results cannot be assessed.

Akbarkhanzadeh (1980) showed that among smokers and non-smokers, mean FVC and FEV_1 were lower in welders than in controls but only the difference in FEV_1 between welders and control non-smokers was statistically significant ($p<0.025$). Results were reported in litres without correction for age and height but there were no significant differences in these variables between groups. Carbon monoxide transfer factor in welders who had never smoked was significantly lower ($p<0.03$) than that of controls who had never smoked.

In a carefully controlled and analyzed study, Keimig et al. (1983) found no consistent differences in FVC and FEV_1, between mild steel welders compared to controls. Values of FEF_{25-75}, FEF_{25}, FEF_{50} and FEF_{75} (forced expiratory flow at 25%, 50% and 75% of vital capacity, respectively) for welders were lower than those for controls but not statistically significantly so. The authors suggested that these decrements in indicators of small airways impairment could suggest an effect of welding which was not statistically significant, perhaps because of 1) the large inter-individual variability of these tests; 2) individuals who were most susceptible to effects of welding fumes could have transferred to other jobs; or 3) welding exposures may not have been of sufficient duration to result in

significant pulmonary impairment. However, correlations between duration of welding employment and spirometric variables were weak. They concluded that a prospective study was needed.

Hayden et al. (1984) reported no significant differences between welders and controls in FEV_1 and peak expiratory flow rate. In a subset of subjects in whom flow-volume curves were also obtained, among smokers, values were significantly lower in welders than controls both for MEF_{25} ($p<0.01$) and for MEF_{50} ($p<0.05$). No differences were found in ex- and non-smokers.

McMillan and Pethybridge (1984) found that FEV_1 declined with age faster in welders than in controls (60 mL/yr vs 40 mL/yr) but these differences were not statistically significant. Residual volume (RV) (33 mL/yr vs 1 mL/yr) and RV/TLC% increased faster in welders, suggesting tendencies to obstructive changes. Using the same system as Hunnicutt et al. (1964), the authors classified lung patterns into normal, obstructive, restrictive or mixed. The prevalence of restriction in welders was greater than that in non-welders, at the 6% significance level. Among non-smokers, six of 23 welders but none of 23 controls had an obstructive pattern. They concluded that there is a suspicion that there may be an excess of obstructive airways disease in welders.

Summary: With the limitations of these cross-sectional studies in mind, the differences in lung function between welders and controls seem fairly consistent. There is limited evidence that these differences in lung function increase with duration of exposure. Kleinfeld et al. (1969) found no such effect and Keimig et al. (1983) reported that correlations between duration of employment and spirometric variables were weak. However, Peters et al. (1973) showed that with increasing years of exposure, RV increased in welders but not in pipecoverers, while TLC dropped in pipecoverers but not in welders. This was compatible with

their prediction if welders suffered obstructive lung disease and pipecoverers did not. Similarly, McMillan and Pethybridge (1984) showed that FEV_1 declined faster and RV increased faster (with age) in welders than in controls. Finally, although the study by Barhad et al. (1975) was not described here in detail due to methodologic selection problems, these investigators demonstrated a trend towards lower FEV_1 (percent predicted) with increasing length of exposure.

One cannot say what process is responsible for these small decrements in lung function although most studies have been in mild steel welders. In the shipyard studies, it is not clear how much of reduced FEV_1, FEF_{25-75}, or peak flow results from welding and how much from asbestos. However, recently presented unpublished data (Kilburn et al. 1985) showed greater symptom prevalences and reduced pulmonary function in young American west coast shipyard welders with little asbestos exposure compared to Michigan males. There has been speculation as to whether recurrent exposure to low concentrations of welding gases such as ozone and nitrogen dioxide causes irreversible impairment of lung function (Parkes, 1982). This speculation has not been resolved.

The probable pulmonary target for inhaled welding fume may be the epithelium of the terminal bronchiole which responds to inhaled particles by developing mucous (goblet) cell hypertrophy (Kilburn et al. 1985). Cross-sectional studies in welders generally report the following decreasing trend of prevalence of impaired respiratory function tests: smoking welders, smoking non-welders, non-smoking welders, non-smoking non-welders, in a possibly additive fashion (Zielhius and Wanders, 1985). Kilburn et al. (1985) suggested an interaction between smoking and welding fume and gas but this needs to be confirmed. Finally, as the incidence of such effects on pulmonary function has not yet been defined and as <u>changes</u> in lung function are more sensitive indicators of adverse effects than actual values, longitudinal (prospective) studies are necessary to clarify the effect of welding on lung function.

4.3 Pathological and Radiological Changes in the Lungs: Siderosis, Mixed Dust Pneumoconiosis and Fibrosis:

Definitions: Pneumoconiosis can be defined as the non-neoplastic reaction of the lungs to inhaled mineral or organic dust and the resultant alteration in their structure excluding asthma, bronchitis and emphysema (Parkes, 1982). This includes harmless as well as potentially harmful changes.

Siderosis is caused by exposure to iron oxide dust such as occurs in electric arc and oxyacetylene welding, producing radiopacities (changes seen on chest x-ray). Siderosis is usally classified among the benign pneumoconioses (Parkes, 1982) as iron oxide is considered to be inert.

Siderosis, due to prolonged pulmonary deposition of iron particles appearing as nodular radiodensities on chest x-rays, is the most commonly occurring form of pneumoconiosis in welders (Palmer, 1983). It was first described in arc welders by Doig and McLaughlin in 1936. The radiographic appearance includes a variable number of small opacities varying from 0.5 mm to about 2 mm in diameter, of striking radiodensity (Parkes, 1982), and have been described as identical to those seen in silicosis (Morgan and Seaton, 1984). Deposition of iron-containing (ferrimagnetic) dust in the lungs can be identified by magnetopneumography as described in Section 1 (Retention and Clearance; Kalliomaki et al., 1980, 1981). Pathologically, the pulmonary pleura in siderosis take on a rust-brown colour due to evenly distributed macules. Microscopically, there are perivascular and peribronchiolar aggregates of dark pigmented iron oxide particles in macrophages, and perivascularly, in alveolar spaces and walls. According to Parkes (1982), there is no pulmonary collagenous fibrosis due to iron alone and, if present, is considered to be due to exposure to other dusts, such as crystalline silica.

The possibility of reversibility (complete or partial clearing) of abnormal x-ray shadows in welders after cessation of, or decrease in, welding exposure was described by Doig and McLaughlin (1948) on follow-up of their original group. Challen (1974) felt that there was little support in the literature for regression of x-ray shadows but noted that few studies had been designed to address the question.

The prevalence of siderosis in the cross-sectional studies reviewed for chronic respiratory symptoms (Table 2) varied from zero in both shipyard (Peters et al. 1973) and non-shipyard (Antti-Poika et al. 1977) welders to 32-34% in shipbuilding (Hunnicutt et al. 1964) and manufacturing (Kleinfeld et al. 1969). A review by Attfield and Ross (1978) noted that prevalence rates up to 80% or more for category 1 had been reported. The prevalence of siderosis was found to be related to duration of exposure, or of employment in welding, by Kleinfield et al. (1969), Ross (1978), Attfield and Ross (1978), but not by Hunnicutt et al. (1964).

Attfield and Ross (1978) have reported the most detailed study of the relationship between the prevalence of small round opacities of class 0/1 or higher (larger than 1 mm "siderosis") and welding exposure based on chest x-rays from 661 British shipyard welders. This included all those currently employed and 12 retirees but it was not stated what proportion of all retirees this represented. Thus no determination of incidence or cumulative incidence is possible. No exposure data were given. No cases of small round opacities 0/1 or greater appeared before 15 years exposure and prevalence then increased with age or with length of exposure reaching over 30% among those with greater than 45 years exposure as shown below for current welders:

Relationship between estimated years of exposure and prevalence of small rounded opacities (0/1 or greater).

Length of Exposure (years)	Age	Prevalence (%) (number of men)	
0-	16-	0	(77)
5-	21-	0	(119)
10-	26-	0	(70)
15-	31-	1.6	(62)
20-	36-	5.7	(53)
25-	41-	11.2	(80)
30-	46-	12.3	(65)
35-	36-	21.6	(51)
40-	56-	18.2	(44)
45-	61-	43.7	(16)
50-	66-	33.3	(12)
All		7.9	(649)

No studies have been reported which would permit a correlation between exposure level to iron oxide and the development of x-ray changes in the lungs (ACGIH, 1980), due to a lack of previous exposure data. For example, Kleinfeld et al. (1969) documented siderosis with concurrent exposures below 10 mg/m^3 but noted that exposures less than 10 years prior to the study had exceeded 30 mg/m^3. A recent report (Goller and Paik, 1985) from an American facility manufacturing locomotive frames found no evidence of siderosis on examination of all 33 welders with an average of 12.5 (range 5-30) years welding. Current exposures to iron oxide averaged 4.7-5.7 mg/m^3 but no historical data were given.

Siderosis has generally been assumed to be benign and unassociated with respiratory symptoms (Morgan and Seaton, 1984) based on the absence of fibrosis both in animals administered iron oxide (Doig and McLaughlin, 1948) and in lungs of welders, and on the absence of pulmonary function abnormalities in welders with marked radiographic abnormalities (Morgan and Kerr, 1963). However, in recent decades, some case reports have suggested that welders may develop siderosis (or some type of parenchymal lung disease) which is not as benign as previously thought. For example,

Charr (1956) described three long-term arc welders who presented with dyspnea and other respiratory symptoms; investigations (including lung biopsy in two subjects), demonstrated impairment of lung function, x-ray opacities and extensive fibrosis. None worked in environments known to contain "a hazardous concentration of silica". Similar reports appeared by Mann and Lecutier (1957), and Friede and Rachow (1961), the latter showing emphysema, fibrosis and siderosis on lung biopsy attributed to prolonged exposure to fumes containing a combination of iron and free silica. Morgan and Kerr (1963) described seven subjects with welders' siderosis, four of whom had lung biopsies. Fibrosis was not seen except in subjects with mixed dust exposures (mixed dust pneumoconiosis). Thus, the possibility was raised that mixed lesions/fibrosis that may complicate siderosis may be associated with silica or other components in welding fume or in the welding environment.

More recent case reports (not all of which will be enumerated) include that by Guidotti et al. (1978) involving a 58 year-old welder who was a smoker with 39 years of welding experience (and purportedly no other known occupational exposures). He demonstrated a bilateral micronodular and coarse interstitial x-ray pattern. On post-mortem, severe interstitial fibrosis was present. Vallyathan et al. (1982) described a case of pulmonary fibrosis in a 35 year-old smoker who had worked for 17 years as an aluminum arc welder, usually in confined spaces in boat contruction. He presented with shortness of breath, mixed restrictive and obstructive pulmonary function tests and had radiological evidence of nodular lesions. Histological examination of a removed lobe demonstrated diffuse fibrosis and dense accumulations of macrophages containing aluminum (identified by energy dispersive x-ray analysis) in the nodular masses and interstitium. The authors were not certain of the role of aluminum oxide in this case.

That clinically significant pulmonary abnormalities can

occur in welders producing symptomatic problems has been discussed by Guidotti et al. (1978) and Loriot et al. (1979). The former group recommended the term arc welders' siderosis to refer to the effects on welders of inhaled iron oxide particles alone and arc welders' pneumoconiosis (AWP) to refer to the effects of inhaling dusts and contaminants generated by welding. They divided AWP into a simple form seen in the majority of cases, without symptoms beyond minimal obstructive airways disease and with a benign natural history; and a complicated form, with unspecified nonferrous dusts and toxic gases taking a major role. Exposure to nitrogen dioxide was named as one recognized hazard among these other constituents.

Stern et al. (1983) reviewed the fibrogenic potential of welding fume, focussing on the 90 or so case reports published which referred to irreversible pulmonary fibrosis in welders, and which were supported by tissue pathology at biopsy or autopsy. In addition to accumulations of particulate material, some of the lung tissue samples from welders revealed evidence of small airway damage and repair (bronchiolitis obliterans) which is not specific for a single agent but can be seen with NO_2 (silo filler's disease). They noted that in only a few of the cases was there a common exposure to stainless steel fume and that the potential contribution of tobacco use could not be examined. They suggested the possible role of NO_2 in such cases noting that Ulfvarson (1981) found that more than 10% of samples from NO_x (about one-half of which was NO_2) exceeded 3 ppm. However, as reviewed in Part I (Gases) the implications for humans of chronic exposure at such levels in the absence of acute intoxicating episodes are not clear. The role of cadmium in producing chronic effects as residua from acute intoxications was described above.

Summary: There is no unifying hypothesis to explain the case reports of fibrosis in welders. No single component in welding fume can be identified as the responsible fibrogen. While the silica present in welding fume, per se,

is recognized to be amorphous, crystalline silica, extraneous to the fume itself, has probably been part of some mixed exposures. Moreover, as there is no estimate for the incidence of pulmonary fibrosis in welders (or for that matter, in the general population), it is not possible to demonstrate if, in fact, an excess of pulmonary fibrosis in welders exists.

4.4 Non-respiratory effects

The fact that attention on health effects in welders has focused on the respiratory system, with the resultant neglect of other aspects, was recently emphasized by Zielhuis and Wanders (1985).

4.4.1 Nervous system effects: Effects on the nervous system have been documented in welders associated with manganese exposure. Although chronic manganism (described in Part I; Particulates) has been reported in other occupations, it has only rarely been found in welders. The epidemiology of manganese intoxication was reviewed by the National Academy of Sciences (1975) in the United States. All articles dealing with industrial exposure referred to miners (not welders) except that by Whitlock et al. (1966) who described two cases of manganism in a small manganese-steel operation. One year prior to the presentation of the cases, a new process for cleaning manganese steel castings containing 10-12% manganese had been introduced in which workers used an electric arc burning operation to burn rough spots off castings. The authors attributed the manganese poisoning to the inhalation of fumes that contained relatively small amounts of manganese.

Subsequent to that review, one of the two cases of manganism reported by Hine and Pasi (1975) in California involved clumsiness in walking, tremor in the extremities, recurrent spells of shaking, speech disturbance, depression and crying spells, and muscular weakness in a 27 year-old welder who had worked for four years in a manganese steel foundry where

his principal task was to burn off metal projections from castings, performed without respiratory protection. There was no local ventilation. Blood manganese levels were normal but an increase in urine manganese was found. An industrial hygiene survey of the work area showed air concentrations of manganese ranging from 2.3 to 4.5 mg/m^3 for welding operations without ventilation and 11.3 mg/m^3 in the arc air operation.

Chandra et al. (1981) examined airborne manganese exposure and neurological signs among welders in India exposed to welding fumes containing manganese. They studied 60 welders (20 each from a well-ventilated equipment fabrication plant, a partially ventilated railway workshop and a ship repair shop where welding occurred in a confined area) and 20 non-exposed controls. Airborne manganese ranged from 0.31 to 1.74 mg/m^3. Positive neurological signs (defined as brisk reflexes or tremors) were observed in 25%, 50% and 45% of the three groups, respectively, and in none of the controls. No cases of manganese intoxication were identified.

4.4.2 Renal Effects: Fatal renal failure has been reported with acute cadmium intoxication in a welder (Beton et al. 1966). Although the kidney is the critical organ in chronic exposure to cadmium, there are no data to evaluate potential chronic nephrotoxicity due to cadmium in welders. Suzuki et al. (1981) noted that among 12 welders working with cadmium-containing silver solder for 3 to 23 years, two were suspected to have suffered renal injury based on urinary total protein, β_2-microglobulin and retinol binding protein but details were not given. Mortality studies in welders have not revealed excess deaths due to genitourinary causes.

Possible associations between stainless steel (chromium) welding exposure and kidney effects have been sought. Mutti et al. (1979) studied (A) 39 stainless steel welders, (B) 36 welders working on armoured steel and (C) 24 chromium

platers in which the groups were distinguished by metal body burden as reflected by chromium clearance and urinary chromium. Indicators used by the authors to detect renal tubular damage included the urinary enzyme beta-glucuronidase, lysozymuria (which, according to Price (1982) is a measure of tubular reabsorption) and proteinuria (which is not specific for tubular damage). β-microglobulin was not determined. The data are summarized in Table 4. Littorin et al. (1984) studied 17 SMA/SS welders, average age 45 years (range 34-64) with an average 20 (range 7-41) years exposure time and referents who were individually matched for age, smoking, alcohol intake, socioeconomic class, living area and drug consumption. The welders had far higher urinary chromium concentrations (median 10.5 vs 0.7 µg/g creatinine). There was no evidence of kidney damage based on the following tests: albumen clearance, urinary β-hexosaminodase, lysozyme and β_2-microglobulin. The first of these is not well established as a parameter of tubular function. β_2-microblobulin and β-hexosaminidase levels were higher in controls but not significantly so. Excretion of β-glucuronidase was not studied.

In summary, the limited studies in welders do not yet permit one to conclude that renal impairment occurs as a result of their occupation. A potential risk (Zielhuis and Wanders, 1985) may exist but further evaluation is required.

4.4.3 Reproductive Effects: There is some evidence that certain nickel and chromium compounds are embryotoxic and teratogenic and for chromium compounds, spermicidal, in experimental animal studies (see Part I; Particulates: chromium).

In Finland, Hemminki and Lindbohm (1985) evaluated spontaneous abortions in female welders and flame cutters and wives of male welders and flame cutters as part of a large study of parental occupational exposure and spontaneous abortion (Lindbohm et al. 1984), using hospital discharges 1973-1976 and occupations from the national

census at the end of 1975. Female welders had a crude rate of spontaneous abortions of 9.5% (age-adjusted 9.7%) which was slightly but not significantly ($p>0.05$) greater than that for other industrial workers (8.5%) or for other employed women (7.7%). There were no differences in rates between wives of welders and flame cutters and wives of other workers.

Rachootin and Olsen (1983) examined associations between infertility or delayed conception and workplace exposures in a Danish case-control study based on hospital records of couples studied for infertility at a hospital during 1977-1980 and control couples who had a healthy child born at the same hospital during 1977-1979. Demographic, occupational and reproductive data were obtained by self-administered mailed questionnaire. There was an increased risk of sperm abnormalities in stainless steel welders [odds ratio 1.7, 95% confidence limits (CL) 1.0-2.8] but not in welders of other metals. This odds ratio remained elevated (1.7) but lost statistical significance (CL 0.9-2.9) after adjustment for women's age, education, residence and number of pregnancies. Delayed conception was significantly associated with welding of other metals in both men (odds ratio 1.3, 95% CL 1.0-1.7) and women (odds ratio 2.7, 95% CL 1.2-6.3) after adjustment. This was not found among welders of stainless steel.

Human studies of reproductive effects in welders are extremely limited, and the preliminary adverse effects reviewed above have been detected in studies not focusing primarily on welders. These reports give no information on the degree of exposure. Detailed human reproductive studies in welders are needed.

4.4.4 Skin Effects: Welding fumes may contain skin sensitizing and irritating substances in addition to the effects of the associated UV radiation and hot metal spatter. There are case reports of contact dermatitis in welders with positive patch test results to potassium

chromate (Shelley, 1964; Zugerman. 1982). Of 250 persons with dermatitis and positive patch tests to potassium chromate identified in a Polish study (Rudzki and Kozlowsla, 1980), 132 had contact with chromates in an occupational setting, and 16 (12%) of these had exposure to welding fumes. It was not possible to determine if the initial sensitization to chromium occurred through non-occupational exposures, through other occupational exposures or through welding exposures.

4.4.5 Lead intoxication: Evidence of excessive lead absorption, non-specific symptoms, presence of lead line, as well as abdominal pain compatible with lead colic has been reported in workers engaged in shipyard and scrap burning and cutting (Rieke et al. 1969). The sources of lead were described in Part I. More recently, Grandjean and Kon (1981) in a survey of active and retired welders in a U.S. ship repair yard, found median blood lead levels of 39 µg/100 ml in 59 active welders and burners; 30 active workers (including 28 welders and burners) had blood lead concentrations above 40 µg/100 ml. Among non-lead exposed populations, blood lead concentrations below about 20 µg/100 ml might be expected.

4.5 Mutagenicity of Welding Fumes

Reports involved both in vitro techniques and in vivo studies. The genotoxicity of nickel and chromium, the components upon which most attention has focused, are briefly reviewed in Part I (Particulates).

4.5.1 In vitro: Hedenstedt et al. (1977) found a mutagenic effect in Salmonella in the Ames system and in Escherichia coli associated with fumes from stainless steel but not mild steel welding. SMA/SS samples initially appeared to produce a stronger mutagenic effect with or without activation by S-9 liver microsomal fraction than GMA/SS (which was mutagenic in only one sample). The components associated with the mutagenic effect appeared to

be water soluble. Similar findings of mutagenicity associated with stainless steel welding fumes in bacteria were reported by Maxild et al. (1978) and Stern et al. (1982).

Hedenstedt et al. (1977) also demonstrated greater frequency of mutations for SMA/SS welding fumes at low concentrations compared to control, in cultured Chinese hamster cells. High concentrations were associated with higher toxicity and poor cell survival.

Koshi et al. (1979) also found stainless steel welding fume to be mutagenic in cultured mammalian cells in vitro. Frequency of sister chromatid exchange (SCE) and chromosomal aberrations increased with increasing fume doses. GMA/SS fume doses 100 times those of SMA/SS were required to produce the same increase in SCE frequency but the amount of chromium in solution from SMA/SS was 60 times that of GMA/SS. It was suggested that dissolved hexavalent chromium may be involved in the cytogenetic effect of the fume particles from stainless steel welding.

Stern et al. (1982, 1983b) presented studies attempting to specify the chemical responsible for the mutagenicity of welding fumes in the S. typhimurium test system. These investigators also felt that there was a strong possibility that the mutagenic activity was partially due to the presence of chromium (VI), an ion with demonstrated mutagenicity. Dose-response data showed that when revertants per plate were plotted against fume mass (mg fume per plate), SMA/SS fume had greater mutagenic activity than GMA/SS. However, when revertants were plotted against µg chromium (VI) per plate, the mutagenicity of GMA/SS was higher than that of SMA/SS and the mutagenicity of the former was found to correspond closely to that of hexavalent chromium as water soluble chromium (VI) in the form of sodium dichromate. Addition of S-9 was found to reduce the observed mutagenicity of various welding fumes and of chromium (VI). This has been attributed to a reduction in

chromium (VI) to chromium (III). Further work by Stern et al. (1983b) showed that the material in GMA/SS fume matrix behaved identically like chromium (VI) from other sources but that the activity of chromium (VI) from SMA/SS matrix is reduced by a factor of 2 to 4 indicating some interference due to the presence of other soluble substances in this more complex matrix. Pedersen et al. (1983) however, cautioned that at high doses of chromium (VI) (as sodium dichromate or from stainless steel welding fumes), a proportion of the colonies thought to be S. typhimurium revertants were "false revertants" because they were unable to grow on histidine-free medium. At low doses, they felt the true mutagenicity of stainless steel fume can be accounted for by the presence of chromium (VI).

The role of nickel in welding fume mutagenicity has not been as extensively studied. The presence of chromium makes the interpretation of the contribution of nickel in stainless steel fume difficult. It should be recalled from Part I (Particulates) that the evidence for genotoxicity of nickel and compounds has to date been considered inadequate by IARC (1982). Pure nickel consumables have been examined. Stern et al. (1982) categorized SMA/MS, SMA/Ni and GMA/Ni as essentially non-mutagenic fumes when examining dose-response data with S. typhimurium. However, Niebuhr et al. (1980) using fume from GMA/MS with nickel rich (95% Ni, 4% Mn) wire showed that the SCE rate in human lymphocytes was proportional to or dependent on the total amount of biologically available nickel. Hansen and Stern (1983) utilized an <u>in vitro</u> bioassay with baby hamster kidney (BHK) cells in culture to assess the relative transforming potential of various nickel compounds at the same degree of toxicity (50% survival). This was presumed to be a direct measure of intracellular concentration of nickel regardless of source. They included an insoluble (presumably carcinogenic) compound Ni_3S_2 (nickel subsulfide), two nickel oxides (Ni_2O_3 and NiO), a soluble salt (nickel acetate) and fume produced by GMA welding with a pure nickel wire. They found that these various nickel compounds were associated

with the same transformation rate for BHK cells at the same degree of toxicity, regardless of source. However, the concentrations and forms of intracellular nickel were not known: neither was the validity of the assumption that survival was, in fact, a measure of intracellular nickel.

4.5.2 In vivo: To assess the effects of welding fume, Knudsen (1980) performed the mammalian spot test which is an in vivo assay for detecting somatic genetic mutations. In this experiment, mutations are induced in melanocyte precursor cells in developing embryos, affecting coat colour loci. Knudsen administered SMA/SS welding fume and two positive controls (potassium chromate and cyclophosphamide) intraperitoneally to pregnant female mice at 8, 9 and 10 days of pregnancy. The welding fume (100 mg/kg), potassium chromate (10 mg/kg) and cyclophosphamide (2.5 mg/kg) produced the same number of spots on the fur of the offspring. The author stated that the effects of welding fume particles may be due to the chromium (VI) content. It should be realized, however, that in this test, germinal mutations are not examined. The limitations of this test include the fact that mutagens must penetrate the placental barrier. Morever, the possibility that some of the colour spots may be due to factors other than genetic alterations at the specific locus involved (e.g. dominant mutations or somatic effects of treatment) cannot be excluded (Fahrig, 1978).

4.5.3 Chromosomal studies in humans: Two studies using peripheral blood lymphocytes from stainless steel (SMA/SS) welders have not demonstrated increases in mutation frequencies compared to referents. Husgafvel-Pursiainen et al. (1982) in Finland assessed chromosomal aberrations and sister chromatid exchanges (SCEs). Littorin et al. (1983) in Sweden considered a number of abnormalities including frequency of cells with breaks and fragments; gaps and isogaps; interchanges and rings; total number of cells with structural aberrations, total number of cells with aberrations; frequency of micronuclei or SCEs. The smoking habits of the subjects were taken into account in both

studies. Possible explanations given by Littorin et al. (1983) for the lack of cytogenetic effect were that SMA/SS welding fume may not, in fact, be mutagenic in man; that the workplace exposures may be too low; or that there may be insufficient distribution of mutagen(s) to lymphocytes. Alternatively, mutagens may reach the lymphocytes but do not generate cytogenic damage. These studies did not exclude the possibility of point mutations.

Summary: Stainless steel welding fumes appear to be mutagenic in bacteria and in cultured mammalian cells. Hexavalent chromium compounds may constitute the active component. No evidence of chromosomal changes has been detected in lymphocytes of welders.

4.6 Carcinogenesis

The welding environment contains a number of known or suspected metal carcinogens including nickel, chromium, cadmium (in brazing), beryllium (in unique situations such as some copper alloys), and arsenic (trace impurity in welding fluxes); of these, nickel and chromium appear in significant concentrations in stainless and alloy steels. The pyrolytic decomposition products from primers, paints and oils on mild steel contain a mixture of organic materials of unknown carcinogenicity. In addition, bystander exposure to carcinogens can occur: for example, approximately 40% of all welders have had some contact with asbestos, often in shipyards (Stern, 1981a).

4.6.1 Non-respiratory cancer: The available studies are tabulated in Table 5.

4.6.1.1 Bladder and kidney cancer: Milham (1976), in an analysis of Washington State death records by the proportional mortality ratio (PMR) method, found a non-significant excess risk for bladder cancer and other urinary organs in welders and flame cutters (observed 12, expected 7, PMR 162). This excess persisted when the analysis was

updated by Milham (1983) (observed 16, expected 12, PMR 128). Howe et al. (1980) performed a Canadian population-based case-control study of bladder cancer which assessed information on occupation in addition to other potential risk factors such as tobacco, coffee and drug consumption. These investigators found an elevated risk among males with exposure to welding fumes [relative risk 2.8, 95% confidence limits (CL) 1.1-8.8] after controlling for smoking. There were insufficient details about the exposure and types of fumes.

Milham (1983) in an update of Washington State mortality, found a PMR for malignant neoplasms of the kidney among welders and flame cutters of 182 based on 20 cases ($p<0.01$). Excesses were found in all three time periods: PMRs were 137, 145 and 234 in 1950-59, 1960-69, 1970-79, respectively.

4.6.1.2 Mesothelioma: Sheers and Coles (1980) evaluated the incidence of mesothelioma occurring during 1966-1978 among shipyard workers at the Devonport Dockyard in Plymouth, England. The seven cases in welders and burners gave a rate for mesothelioma of 80 per 100,000 man-years compared to 206, 161 and 119 in laggers and asbestos insulation sprayers, boilermakers and painters, respectively, while in asbestos-unexposed workers, the rate was 16 per 100,000 man-years. An increased incidence of mesothelioma in welders (relative risk 3.4) was also found by Sjogren et al. (1982), again suggesting exposure to asbestos.

4.6.1.3 Larynx: Olsen et al. (1984) examined the occupational histories of laryngeal cancer patients, focusing on exposure to welding. Cases were all newly diagnosed patients in Denmark 1980-1982; age- and sex-matched controls were selected from the municipal registers in which cases were listed. Overall, welders had an elevated risk of laryngeal cancer (relative risk 1.6, 95% CL 1.0-2.4 by matched analysis), adjusted for age, alcohol

intake and smoking: for the subtype of subglottic cancer, the relative risk was 8.1 (95% CL 1.5-43.3). The increased risk was confined to smokers but the numbers of non-smokers was small. A similar pattern was observed among the smaller sub-group with stainless steel welding but gave imprecise estimates.

4.6.1.4 Nasal and sinonasal cancer: Hernberg et al. (1983) reported the results of a case-control study examing the relationship between occupational exposure and nasal and sinonasal cancer. All new cases of these cancers were collected from registries (in Finland and Sweden) or from hospitals (in Denmark). Those who were still alive and who agreed to an interview were matched for age and sex with patients with colonic or rectal cancer. There was an association of nasal and sinonasal cancer with the exposure category "welding, flame-cutting and soldering" (odds ratio 2.8, 95% CL 1.1-9.4). Of the 21 welding cases, exposure to chromium was reported by 12, and to nickel by 11 individuals.

4.6.1.5 Skin Cancer: Because of potential exposure to UV radiation, increased risk of premalignant and malignant skin disease might exist in welders but no comprehensive studies have examined this (Zielhius and Wanders, 1985). Emmett et al. (1981) examined, among other things, the prevalence of skin cancer in a cross-sectional study (limited to Caucasians) of 77 journeymen welders employed at a large metal fabrication facility for more than 10 years and a comparison group of 56 employees who had never worked as welders but had worked at the plant for at least 10 years. No significant differences in the prevalence of skin tumours was observed.

Further work will be required to confirm these associations of non-respiratory cancer and welding. If confirmed, the burden of illness due to those associations, however, would be small compared to a definitive increase in respiratory cancer.

4.6.2 Respiratory cancer

Because of the presence of potential carcinogens in welding fume, and the respirable nature of the fume, it is to be expected that attention should be paid to the risks of respiratory cancer in welders. The epidemiological studies available to assess associations between respiratory cancer and welders (Tables 6 and 7), may be categorized into: 1) studies based on all deaths or cancer registrations in a country or region; 2) studies of shipyard welders; and 3) studies of non-shipyard welders. Within the latter two groups, studies can be ranked in quality of study design (cohort, case-control and so on). Most were not designed primarily to study welders

1. Studies based on occupation recorded on death certificates and/or census returns. Based on deaths during 1968-69 and the decennial supplement from the Office of Population Censuses and Surveys, an SMR for lung cancer of 151 ($p<0.05$) was found for men aged 15 - 64, in English and Welsh gas and electric welders, cutters and braziers, (Registrar General, 1978).

Menck and Henderson (1976) studied all 2,161 lung cancer deaths by death certificate (1968-70) and all 1,777 incident cases (1972-73) in white males in Los Angeles County. These data were then combined with occupation and industry information from a 1 in 50 sample of the white males in the same county. The estimated population of welders was 15,300. There was a total of 48 lung cancer deaths in welders (SMR 137, $p<0.05$). No information on previous occupation, asbestos exposure or smoking was available.

Gottleib (1980) performed a case-control study of lung cancer in Louisiana in 19 parishes selected because of their high lung cancer death rates or because of their concentration of petroleum, chemical and paper manufacturing industries. Lung cancer cases were identified from all Louisiana deaths and an equal number of non-cancer deaths

were selected as controls. These were then matched to the cases by sex, race, year of death, parish of residence at death and age at death. Occupation and industry information was extracted from the death certificates. Eight cases versus two controls were welders (odds ratio 4.01).

Milham (1983) in the update of Washington State mortality data, found a PMR of 1.35 for lung cancer in welders based on 153 cases ($p<0.01$). Excesses of similar magnitude were observed in all three time periods: 1950-59, 1960-69, 1970-79. Excess deaths were also observed due to chronic bronchitis (PMR 256, $p<0.01$) based on 14 cases, which may reflect a smoking effect.

However, since the occupation data in these studies are based on information recorded on death certificates or from censuses (i.e. not individuals), the validity of the findings is uncertain. However, Sjogren et al. (1982) matched 26,000 welders and gas-cutters identified in the 1960 Swedish census against the National Cancer Registry for cancer development during 1961-1973 in a true follow-up study. Compared to the number of cases expected based on the experience of the total population, the standardized incidence ratio for lung cancer was 1.44 ($p<0.001$).

It is noteworthy that the estimates from these studies are similar. The lung cancer death or incidence rate among men identified with usual occupation of welder appeared to be about 40% above that of the general population. Smoking habits were not considered in these studies.

2. Studies based on shipyard welders: Table 7 summarizes the studies based on shipyard welders, divided into cohort and case-control reports. Puntoni et al. (1979) examined mortality patterns in shipyard workers in Genoa who were working, had retired or were dismissed in 1960-1975. Among 214 welders (3829 man-years), 7 lung, bronchus and trachea cancer deaths were observed compared with 5.08 expected (SMR 1.38) based on the age-specific death rates of the male

population of Genoa. No information on asbestos exposure or tobacco use was available.

Beaumont and Weiss (1981) performed the only study of shipyard welders with enough lung cancer deaths to provide a precise estimate of the relative risk. The vital status of 3247 welders who had been members of a Seattle union local for at least three years between 1950 and 1973 was determined as of 1977. External comparisons were made using the age- and time-specific mortality rates for U.S. white males. Internal comparisons of lung cancer rates were made between the welders and 5432 non-welders of the same local. Analysis of lung cancer by duration of exposure and latency period were also performed. The vital status of all but 2% of the welders cohort was known. There were 50 deaths due to lung cancer vs 37.95 expected based on U.S. rates (SMR 132, $p=0.06$). When only deaths occurring 20 or more years after first employment were considered, there were 39 lung cancer deaths vs 22.38 expected (SMR = 174, $p<0.001$). The SMR was found to increase with latency category but not duration of exposure (Figure 2). No similar increase with latency occurred among non-welders who likely had similar smoking habits. The directly standardized relative risk based on comparison with non-welders was 1.28 (95% CL 0.89-1.84).

Obviously these studies are difficult to interpret because of the small size of all the studies except that by Beaumont and Weiss (1981) and the probable exposure of the populations to other occupational carcinogens including asbestos. However, some of the relative risk estimates among shipyard welders are similar to the estimates from the population studies above. The duration of welding work was not well characterized in either group of studies.

3. Studies based on non-shipyard welders: These reports are summarized in Table 7, again stratified by study design. Although these studies should be the most informative as to magnitude of risk with respect to welding

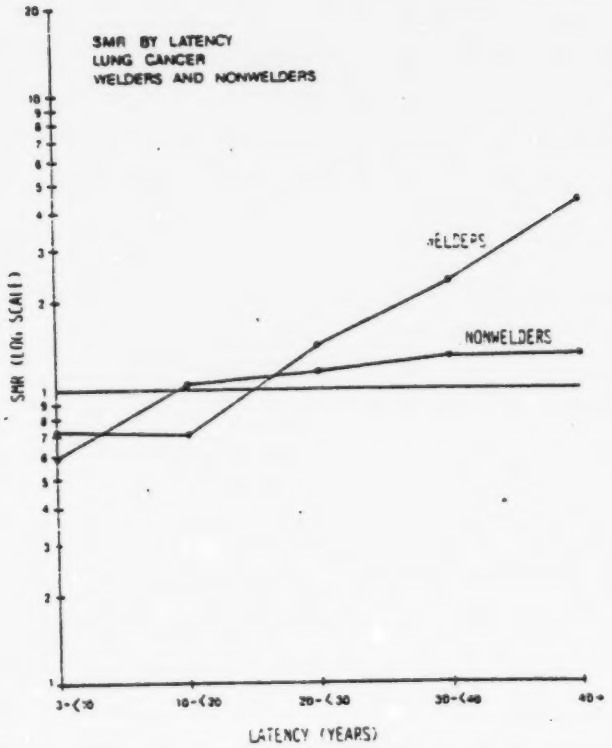

Fig. 2 - Lung cancer in welders and nonwelders in Local 104: standardized mortality ratios by latency where first exposure is entry to the union.

(from Beaumont and Weiss, 1981)

type or duration of exposure, they usually involved small numbers of subjects or lacked detailed analysis.
The earliest cohort study (Dunn and Weir, 1968) was part of a prospective study of mortality (with emphasis on lung cancer) of several Californian occupational groups. These groups were identified in 1954-1957 and followed up by checking all adult male deaths in California during 1954-1962 against the names of the study populations. Many of the occupations of interest were identified as a result of a case-control study involving occupations and lung cancer (Breslow et al. 1954) also included in Table 7. The total study population (all occupations) totaled 68,153 men with 482,658 person-years of observation and the expected number of deaths was calculated on age- and smoking-specific rates for the total study population. Among welders, there were 81,389 person-years of observations for individuals aged between 35-69 years, and for lung cancer, there were 49 observed deaths vs 46.5 expected (relative risk 1.05). However, the expected number may have been overestimated because of the smoking corrections used.

Ott et al. (1976) reported on a cohort of Midland, Michigan chemical workers employed from 1950 to 1954 and followed to 1972. Comparisons were made with the U.S. population (indirect standardization). There were 180 welders and leadburners among the total 5994 men followed, and among welders, there were 12 deaths due to malignancy (7.4 expected). Two deaths due to respiratory system cancer occurred which was approximately the number expected statistically.

Redmond et al. (1979) examined the mortality experience of welders as part of their large study of Pennsylvania steelworkers. They followed 644 white welders for deaths during the period 1953-1975. Compared to the U.S. population, the SMR for lung cancer in this group was 151 (14 deaths, 9.28 expected, not statistically significant).

Sjogren (1980) reported what was until 1985, the only published study to date that followed a cohort of welders of

stainless steel. The cohort, assembled from lists of welders provided by eight companies in Sweden, involved 234 male welders who had stainless steel welding as their main task for at least five years between 1950 and 1965. Mortality through the end of 1977 was traced for 100% of the cohort through the registration books of local parishes, the Swedish National Central Bureau of Statistics (SNCBS) and the National Cancer Registry. The expected numbers of deaths were calculated based on age-, sex-, calendar time-, and cause-specific rates given by the SNCBS. Person-years at risk were calculated after the minimum five-year exposure time. Exposure in stainless steel welding was surveyed in Sweden in 1975. For welding with covered electrodes, the median time-weighted average exposure to chromium in 1975 was 210 $\mu g/m^3$; the highest concentration was 1,440 $\mu g/m^3$ and 85% of the available 85 measurements for chromium exceeded 50 $\mu g/m^3$. For GMAW, the median chromium concentration was 20 $\mu g/m^3$, the highest 840 $\mu g/m^3$ and about one-third of 41 chromium measurements exceeded 50 $\mu g/m^3$. There were no measurements available for 1950-1975. There were 17 observed deaths (expected 17.75) for all causes, four deaths (expected 4.01) for all tumours, and three welders died from pulmonary tumours (expected 0.68, SMR 441, p=0.03). Durations of exposure were 17, 17 and 29 years for the three cases. However, no analysis by duration of exposure or latency for this small group was reported. The frequency of smoking among Swedish welders was not considered to differ importantly from that of the Swedish male population.

Polednak (1981) examined the mortality of all welders who had worked at three Oak Ridge, Tennessee nuclear facilities between 1943-1973. The study end date was January 1, 1974. The 1059 white male welders included two sub-groups: 536 that had worked on nickel-alloy pipes (mild steel with nickel coating at plant K-25) and 523 that had used GTAW and SMAW on mild steel, stainless steel, and aluminium. Follow-up was completed for about 92% of the cohort and the expected numbers of deaths were generated using U.S. white male age- and calendar time-specific rates. Time-weighted

average concentrations obtained in 1975-1977, ranged from 0.04 to 0.57 mg/m^3 for nickel, and from 0.18 to 0.47 mg/m^3 for iron depending on the welding process. Smoking histories were available for about one-third of the welders. The proportion of "heavy smokers" in K-25 was similar to that in the U.S. population. There was a non-significant excess of deaths due to lung cancer (observed 17, expected 11.4, SMR 150) and rates at the two plants were similar. Overall, there was little difference in relative risk between men employed for under or over 50 weeks. However, duration of employment solely in the workplace under study may be unrelated to lifetime exposure, as welders may work elsewhere before joining and after leaving. At plant K-25, the SMR for respiratory cancer was higher in welders employed for 50 or more weeks (SMR 175, CL 57-408) based on only 5 cases, than in those employed less than 50 weeks. The cohort was diluted with individuals who worked for only short periods as welders. The influence of latency was assessed among 922 welders with at least 15 years of follow-up; 16 lung cancers were observed (9.1 expected, SMR 176) which was slightly greater than that observed overall. Wong et al. (1983) noted that this SMR was on the borderline of significance at the 0.05 level (95% CL 100.4 - 285.5). Polednak concluded that an increased risk of respiratory tumours following nickel exposure could not be excluded.

Becker et al. (1985) recently reported the findings of a retrospective follow-up study of chromium- and nickel-exposed welders in the Federal Republic of Germany, the only such study besides that of Sjogren (1980). The study cohort included 1224 arc welders of high alloy chromium-nickel steel from 25 factories (manufacturers of sanitary installations, power plants and boilers) employed since the 1950s and first exposed before 1970. An internal reference group defined from personnel files consisted of 1694 turners, milling cutters and drillers (referred to as "turners") recruited from most of the 25 factories and also employed since 1950. Data on smoking habits were collected by interviews with foremen. Details concerning

electrodes and processes were also ascertained. For external comparison, mortality in the two groups was indirectly compared to cause-specific mortality rates of the Federal Republic of Germany for males from 1952 to 1978. Only 2-3% of the cohort was lost to follow-up, and death certificates were available for 96.3% of the deaths. For the internal comparison, results with stratification by smoking status showed no signficiant differences in rate ratios compared to a simple evaluation; smoking was not considered further in the analyses. Compared to the turners, there was a statistically significant increased risk from deaths due to (all) malignant neoplasms in the welders (rate ratio 2.4, 95% CL 1.1-5.1). Of note, two cases of mesothelioma were observed among welders although none were found among turners (for one, a former occupational exposure to asbestos could be confirmed). The rate ratio for cancers of trachea, bronchus and lung was 1.7 but this excess in welders was not statistically significant (95% CL 0.7-4.0). External comparison showed that fewer deaths were observed than expected for all causes, for all malignant neoplasms and for lung cancer, in both welders and turners. For lung cancer, the SMR in welders was 95.4 (6 observed, 6.3 expected), and in turners was 69.2 (10 observed, 14.5 expected). However, in welders, the SMR for lung cancer increased with increasing time since first exposure, reaching 163 for 20-29 years and 175.6 for 30 years: no such trend was observed in turners. For the different causes of death, SMRs, although mostly below 100, were always higher for welders using coated electrodes than for welders using other techniques. The authors concluded that their study indicated an increased cancer risk for arc welders of high alloy chromium-nickel steel. However, due to the cohort size and the short observation period, a definite statement regarding individual cancer sites could not be made.

The first case-control study available was that by Breslow et al. (1954) undertaken in part to determine whether different occupations might be associated with different

lung cancer rates. Cases were lung cancers occurring in 11 California hospitals between 1949 and 1952. The controls were patients matched for age, sex and race who were in the same hospitals at the same time for a condition other than cancer or chest condition. Interviewers obtained information on occupation and smoking. Welders had an increased risk of lung cancer (14 welders, 2 controls, odds ratio 7.2 uncorrected for smoking status; the odds ratio remained elevated after adjustment for smoking). However, it is not possible to have much confidence in this estimate of relative risk due to the small numbers. This study was not designed specifically to study welders.

Gerin et al. (1984) explored associations of nickel and cancer as part of a population-based case-referent study of 12 tumour types and occupational exposure. The study recruited incident cancer cases in the entire Montreal population of males aged 35 to 70. Each incident case was interviewed for sociodemographic variables and detailed job history. Each job history was evaluated by chemists who inferred what the possible physical and chemical exposures were. Comparisons were carried out for each cancer site with a referent group consisting of all other interviewees. Only the nickel-lung associations were positive. The only specific occupation/industries in which nickel exposure occurred that showed a positive association with lung cancer was the class "welding and flame cutting occupations", with 12 exposed lung cancer cases (odds ratio 2.4 95% CL 1.0-5.4). Welders with nickel exposure (mostly stainless steel welders) had a high risk of lung cancer (odds ratio 3.3) while welders without nickel exposure had little or no excess risk (odds ratio 1.2 but based on only two exposed cases with lung cancer). These analyses involved Mantel-Haenszel estimates with stratification based on age, ethnic group, socioeconomic status and amount of cigarette smoking. Analyses for the exposure "chromium and compounds" showed similar results. As exposure to chromium compounds was highly correlated with nickel, the observed nickel associations may have reflected a confounding effect of

chromium (Gerin et al. 1984).

The interpretation of the welding-lung cancer association which appears to exist must take into account cigarette smoking, which probably can explain, in part, differences in lung cancer incidence between countries or within countries at different times. To be a confounding factor, smoking must be associated with the exposure (i.e. welding) in addition to being a risk indicator of the disease. The General Household Survey (Registrar General, 1978) which gave data on smokers in England and Wales, 1972-73, indicated that among welders, estimates of the proportion of current smokers were 22% higher than expected. This could account for about 37% excess risk for welders in England and Wales (Stern, 1983b). It is not known if this smoking estimate applies to other welding groups although among Swedish welders the frequency of smoking was not considered to differ from that of the Swedish male population (Sjogren, 1980). It has been suggested by Doll (1984) that there is little difficulty in recognizing an occupational hazard when the relative risk is greater than two but may be extremely difficult if it is much less.

Summary: The results in Tables 6 and 7 are generally consistent with a relative risk for lung cancer of the order of 1.3 or 1.4 (Peto, 1985) and are based largely on mild steel welders. This has also been suggested by other reviewers (Stern, 1983a,b; Beaumont and Weiss, 1981). There are several possible explanations for this increase which are summarized in the following paragraphs. The first possibility is that the risk is negligible and the confounding effects of smoking (and perhaps exposure to other occupational carcinogens) could account for the results.

The second possible explanation is that mild steel involves little or no risk but that exposure to certain welding processes in particular stainless steel, constitutes a "hot spot" (i.e. a higher risk of lung cancer; Stern, 1983a,b).

In this explanation, the increased risk in welders can be explained by some excess smoking, some exposure to asbestos and the small (10%) of welders exposed to chromates in stainless steel welding where there might be, perhaps, a three-fold relative risk as suggested by Sjogren (1980). This theory also considers that stainless steel welding fumes contain hexavalent chromium, that stainless steel welders absorb chromium, that stainless steel welding fumes contain mutagenic compounds, that chromium (VI) appears to be the (an) active contributor to this mutagenicity, and that there are recognized increased relative risks for lung cancer in other industries where workers are exposed to chromates. Drawing on the data from these non-welding exposures, Stern (1983a,b) estimated that the relative risk may be approximately 6 among stainless steel welders with a cumulative exposure to chromium of about 3 (mg/m^3 years). Stern (1983a) noted that there was no evidence to support the premise that exposure to nickel and/or chromium in welding fumes present risks which are different from exposures resulting in equivalent doses in non-welding industries. Although there is not yet sufficient epidemiological or experimental evidence that this "equivalence of risk" from industry to industry applies to welders, the fact remains that this sub-group is exposed to hexavalent chromium. The only human reports available are those by Sjogren (1980) in which a relative risk of 4.4 was found and that by Becker et al. (1985) in which no increase in lung cancer was observed on external comparison, but a rate ratio of 1.7 was noted on internal comprison with non-welders in the same factories. However, these studies were very small (three and six cases, respectively) and need to be confirmed. Any potential risk from nickel appears negligible compared to that from chromium (VI) (Stern 1983a).

The third possible explanation is that long-term employment as a welder of any type may cause a substantial increase in lung cancer after a sufficient latency period has elapsed (e.g. over 20 years). The study by Beaumont and Weiss

(1981), the only one that has been analyzed by duration of employment and latency, showed an overall relative risk of 1.3 (which is similar to other studies) but 1.74 after 20 or more years from first exposure ($p<0.001$) (Figure 2). However, the number of lung cancers observed beyond 30 years was not stated but was probably small and so the results could be due to chance. The fact the risk of cancer bore no relationship to the duration of exposure in this study does not constitute strong evidence against welding as a causal agent because duration of membership in the union or employment with a company does not exclude exposure by a member of a skilled trade before or after belonging to the union or the company. Moreover, Doll (1984) described studies of occupational lung carcinogens that have shown weak or inconsistent relationships with duration of exposure (e.g. beryllium and asbestos). There is as yet no candidate carcinogen in mild steel identified as part of this explanation and mild steel fumes have not been shown to be mutagenic.

Considering the quality of the available historical prospective cohort mortality studies of welders, the size of most studies was extremely limited and quantification of exposure was generally unavailable (industrial hygiene data were reported for only two studies). No authors have attempted, based on the data of their studies, to relate cumulative exposure or dose to risk. Confounding factors could not be properly considered: only the presence or absence of shipyard work is known and, as with most occupational mortality studies smoking histories were not obtained. Ascertainment of mortality was generally adequate although the proportions of person-years-at-risk falling into various lengths of follow-up categories are not available. Analysis with allowance for latency effects was not attempted in any study except those by Beaumont and Weiss (1981), Polednak (1981) and Becker et al. (1985).

In evaluating whether the association between welding and cancer is causal in nature, the modified Bradford Hill criteria incorporated in Advisory Memorandum 82-V (ACOHOS, 1983) can be considered. The association of lung cancer and welding appears consistently from study to study and has a correct temporal relationship. However, the association is not strong, although possible effects in the existing studies may be diluted by mixed exposures and processes, and by inclusion of welders with short duration of exposure. A dose-response gradient (considering duration of exposure) has not been demonstrated but possible explanations for this were discussed above and by Doll (1984). The "hot spot" theory involving hexavalent chromium exposure makes biologic sense, and is consistent with the epidemiologic evidence in other industries with chromate exposure.

Peto (1985) noted "we are in the curious position of not knowing whether welding is among the most important current occupational carcinogens or whether its effects are negligible." One cannot dismiss the data accumulated to date. The need to assemble large cohorts of welders consisting of groups with long duration of exposure to stainless and mild steel (greater than 10 years or even lifetime) and followed beyond 20 years from first exposure, has been emphasized. The published studies do not note the proportion of observations falling into this category but it is likely to be quite small. The available studies provide inadequate evidence for assessment of the carcinogenicity of welding fume in man. An international collaborative study to address this question has been proposed (W.H.O., 1984).

CONCLUSION

In the welding environment, there is potential for exposure to a number of substances, the individual toxicities of which have been fairly well documented in non-welding, and in some cases, welding settings. Data on the effects of multiple chemical exposures is generally lacking but are not necessarily additive (Ballantyne, 1985). With respect to

acute effects, information is available mostly from case reports attesting to acute (respiratory) health effects in welders. These include metal fume fever, excess mortality due to pneumonia, and upper respiratory irritation due to contaminants such as ozone and fluorides. In addition, pneumonitis/intoxication has occurred, generally restricted to process-dependent exposures such as ozone, oxides of nitrogen or cadmium, due to work in confined or poorly ventilated spaces or as a result of work with lack of knowledge of content of filler materials or surface coatings.

With respect to chronic health effects in welders, data is more limited or non-existent. The available epidemiologic studies of respiratory effects based on cross-sectional design suggest that welders have excess prevalence rates for symptoms of chronic bronchitis and possibly of reduced pulmonary function. Longitudinal investigations will be necessary to measure the magnitude of the risk for the decrements and to relate them to specific processes. Although there has not been sufficient focus to date on non-respiratory health effects in welders (Zielhius and Wanders, 1985), welders appeared to demonstrate increased absenteeism and skin diseases (including allergic contact dermatitis), while there are tentative findings related to the reproductive system such as increased rates of spontaneous abortions in female welders and flame cutters and delayed conception associated with welding in Scandinavian studies. However, this and other systems will require further study. Cases of additional chronic illness related to, for example, beryllium (chronic beryllium disease) and manganese (neurological disorders) have been rarely associated with the exposure to these toxic contaminants from specific welding consumables. There is consistent evidence for a weak association of lung cancer with welding from existing studies but these studies have methodologic limitations (small size, confounding exposures, mixed processes, absence of hygiene data for study populations, absence of analysis by duration of exposure or by

latency). Large cohorts of long duration/long latency subjects will be needed to clarify whether there is a causal relationship between welding and the development of lung cancer.

Taken as a whole, there is reason for concern that prolonged exposure to concentrations of welding fume that existed under past and perhaps current conditions may present some degree of hazard to the health of welders. While the welding environment often contains known toxic elements and welders can be shown in some studies to suffer chronic health effects, there is inadequate data allowing the magnitude of the exposure to be related to the health effects, much less to levels of specific contaminants which can be deemed safe or not safe.

Apart from the epidemiological information, there are documented cases of exposures of individual welders to total fume and to specific (mostly particulate metal) constituents, in excess of current guidelines and threshold limit values, in Ontario and elsewhere (Jenkins et al. 1981). The potential for significant process-dependent exposures (such as to chromium or to ozone) has been emphasized.

As might be expected as a corollary to the absence of dose-response relationships for health effects in welders, the documentation for recommended or enforceable exposure criteria for welding fumes in various jurisdictions has been based on limiting exposure (i.e. the inherent potential health effects attributable to specific fume constituents) and not on relationships to chronic health effects demonstrated to occur in welders at specific levels of welding fume exposure. For example, recommendations have been made for total fume for unalloyed metals (5 mg/m^3, ACGIH 1980; Health and Safety Executive, 1984) with reference to individual TLVs where more toxic constituents are present (as recommended by ACGIH). On the other hand in Denmark in April 1984, process-dependent TLVs were

promulgated for five process/base metal combinations, listed below as an example solely for illustrative purposes (Beck Hansen, 1985; Direktoratet for arbejdstilsynet, 1984):

Process	Base Metal	TLV (mg/m^3)
SMAW	unalloyed steel	3.4
GMAW	unalloyed steel	3.1
Flame cutting	unalloyed steel	2.8
GTAW	stainless steel	2.7
SMAW	stainless steel	1.6

These process-dependent TLVs were developed from a statistical analysis of a data base at the Danish Welding Insitute consisting of approximately 1400 air sampling results for different processes, filler materials, base materials and so on. The resulting TLVs (TLV_{res}) above were calculated from the formula:

$$TLV_{Res} = 100/(A_1/TLV_1 + A_2/TLV_2 + \cdots + A_R/TLV_R)\ mg/m^3$$

where

A_X = the content of an individual element of the fumes in %;

TLV_X = the corresponding threshold limit value of the element in mg/m^3;

A_R = the remaining fumes, i.e. the part of the fumes not identified by analysis;

TLV_R = the threshold limit value for the remaining fumes = $5mg/m^3$.

Such a system has the advantages of simplifying the method of assessment (only a gravimetric rather than multiple

elemental analysis is required); reducing cost; including non-ferrous constitutents even with mild steel; and providing a guideline or "ballpark" to industrial hygienists for all processes/consumables. However, a limitation of this scheme is its dependence on the additivity of component TLVs to form a "resulting TLV". There is no physiologic basis for this, especially since the constituents may act by different mechanisms.

It is apparent that in most cases, either a single element dominates the determination of an acceptable fume concentration level (as in stainless steel) or, for unalloyed steels, toxic elements are probably not present in sufficient quantities to significantly reduce allowable levels to below 5 mg/m^3 (Tinkler and Graville, 1983). It should be re-emphasized that with few exceptions (such as chromium in SMA/SS, welding of copper alloys, gas-shielded welding of aluminum and stainless steel where ozone is a problem, and perhaps fluorides, barium and beryllium), the majority of exposures to individual elements do not generally exceed their respective TLV values provided the TLV for total fume is not exceeded (Stern, 1981a - assuming 5 mg/m^3 for total fume; Jenkins, 1981). Despite the absence of dose-response relationships, in view of the potential and demonstrated health effects, the exposures of welders should be limited.

RECOMMENDATION

The recommendation is that exposure to welding fume should be controlled perhaps by adjustments directed at parameters of the welding process (substitution where possible, control of length of arc, power, electrode core diameter); by ventilation guidelines based on process, and base metal and consumables; by limiting exposure to total dust (mild steel welding fume) to 5 mg/m^3; or by some combination of these approaches.

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Appendix I

Approximate Ranges of Exposure to Specific Welding Contaminants

Contaminant (TLV)	Reference	Process	Conditions	Exposure
Ozone				
(0.1 ppm)	Lunan (1967)	SMAW) on MS FCAW) GTAW on MS,SS,AL	Natural ventilation	Mean <0.1 ppm
		Bare-wire argon-shielded (GMAW) on SS on pure Al on Al-Mg	Natural ventilation	Mean 0.35 ppm Mean 0.47 ppm Mean 0.25 ppm
	Ulfvarson (1981)	GMAW on Al	Inside face guard	Mean just below 0.1 ppm (90th percentile 0.4 ppm)
Oxides of nitrogen				
(NO_2 3ppm NO 25 ppm)				
	Morley and Silk (1970)	Flame cutting	Area (NO_2) samples	All above 5 ppm (Range 7.7 - 16.5 ppm)
		Arc welding	Confined/ semi-confined spaces (NO_2)	Mean 7.4 ppm (Range 4-20 ppm)
			Open air/ open workshop (NO_2)	Mean 1.0 ppm (Range 0-2.0 ppm)
	Ulfvarson (1981)	GTAW on Al	Outside face guard (NO_x)	Mean 1.0 ppm (90th percentile 4 ppm)
		GMAW on Al	Outside face guard (NO_x)	Mean 0.5 ppm (90th percentile < 3 ppm)

Appendix I (continued)

Chromium:

Stainless steel welding fumes are characterized by the presence of high concentration of chromium and nickel in different oxidation states and solubility fractions.

In SMA/SS, almost all chromium is in the soluble hexavalent form. Ulfvarson (1981) found during sampling that most determinations exceeded 0.02 mg/m^3 (20µg/m^3) while with GMA/SS, only a portion was water soluble and levels were lower. Stern (1981a) reported that high alloy and stainless steel fume contained 2-13% chromium, while for mild steel, the chromium fraction was less than 0.1%. The proportion of hexavalent chromium in fume varies with the process, making up 2-4% in SMA/SS (almost all water soluble) but only 0.02-0.05% of GMA/SS (both soluble and insoluble). However, these differences could be minimized by higher duty cycles and smaller particle size for GMAW.

Chromium (VI)			
Stern (1981a)	SMA/SS	Experimental (standard fume box conditions)	150-300 µg/m^3
	GMA/SS	Experimental (standard fume box conditions)	35 µg/m^3

Tinkler and Graville (1983) showed that in SMA/SS welding, using an example of 5% chromium content, 70% of which is hexavalent (3.5% of total fume), a total fume exposure of 5 mg/m^3 would be associated with an exposure level of 0.175 mg/m^3 (175 µg/m^3) hexavalent chromium. Total fume below 1.43 mg/m^3 would be necessary to achieve a Cr(VI)

Appendix I (continued)

concentration below 0.05 mg/m^3. Similarly, considering the proportions of insoluble and soluble Cr(VI) in GMA/SS welding, a total fume exposure of 3.5 mg/m^3 would result in total chromium levels below 0.5 mg/m^3 while hexavalent chromium would be below 0.05 mg/m^3 with fume level below 10 mg/m^3 (thus, total chromium would be the limiting factor).

Nickel:	Ulfvarson (1981)	Stainless Steel	Behind Face Shield	Commonly below 0.1 mg/m^3 (100 µg/m^3)
	Stern (1981a)	SMA/SS	Standard fume box (no shield)	50 µg/m^3
		GMA/SS		250 µg/m^3

GMA welding with nickel consumables typical for cast iron can produce nickel fractions of 40-60%, primarily of highly crystalline Ni and NiO. Tinkler and Graville (1983) stated that it was unlikely that "excessive" (greater than ACGIH TLV) nickel exposures would occur if total fume of 5 mg/m^3 is maintained.

Copper: Tinkler and Graville (1983) noted that during use of GMA copper clad filler wire, it was likely that total fume would exceed 5 mg/m^3 before copper fume TLV of 0.2 mg/m^3 was exceeded (i.e. copper would not be a limiting factor).

Fluorides: With the use of basic (low hydrogen) electrodes and FCAW or SMAW, fluoride can compose up to 35% of the fume and exposures above the recommended level of 2.5 mg/m^3 are not uncommon.

Appendix II

Checklist for Epidemiological Studies of Respiratory Symptoms and Pulmonary Function

1. Are subjects representative of population from which drawn is chosen without selection bias?

2. Is there a control group?

3. Is there adequate matching of study and control groups for age and sex?

4. Is there control for socioeconomic status?

5. Is there control for smoking?

6. Is there control of other confounders (e.g. asbestos)?

7 Was spirometry performed adequately?

8. Was questionnaire for symptoms adequate?

9. Were interviewers blind to exposure status during assessment and evaluation?

10. Are appropriate analysis and statistical tests applied? e.g. separate analysis of smoking groups?

Appendix II (continued)

Study (Yr)	Subjects Representative	Matching Age & Sex	Control S.E.S.	Control Smoking	Control Other Confounders	Questionnaire Adequate	PFTs Adequate	Blinding of Observers	Appropriate Analysis
Schuler (1962)	No	No controls							
Morgan (1963)	No	No controls							
Hunnicutt (1963)	Yes	Frequency matched	Yes	Yes	Partially	UNSP	UNSP	UNSP	Yes
Stanescu (1967)	No (just siderotics)	Frequency matched	UNSP Matched	Frequency	"No Dust Exposure"	UNSP	UNSP	UNSP	No: combined analysis of smoking groups only
Kleinfeld (1969)	UNSP	Yes	UNSP	Yes	"No Occup Dust Exposure"	UNSP	Probably	UNSP	No: combined analysis only
Fogh (1969)	UNSP (Part of controls randomly selected)	Yes	Yes	Yes	"Sections of plant where welding not normally done"	Yes	UNSP	UNSP	No: combined analysis for symptoms
Peters (1973)	Yes	Yes	Yes	Yes groups	Yes with additional control	Yes	Yes	UNSP	Partial-combined analysis with additional controls
Ulrich (1974)	UNSP	Yes	Yes	Yes	Yes	UNSP	UNSP	UNSP	Yes for symptoms (PFT inadequate)
Barhad (1975)	Cannot tell (subjects & referents with chronic pulmonary disease excluded)		Not further considered						

Appendix II (continued)

Study (Yr)	Subjects Representative	Matching Age & Sex	Control S.E.S.	Control Smoking	Control Other Confounders	Questionnaire Adequate	PFTs Adequate	Blinding of Observers	Appropriate Analysis
Antti-Poika (1977)	UNSP	Frequency matched	Yes	Frequency matched	38% of welders 16% of controls exposed to other dusts	Yes	No correction for ht & age	UNSP	No separate analysis for smoking groups
Ross (1978)	UNSP	Yes	Yes	Yes	Yes (attempted)	UNSP	UNSP	UNSP	Yes (but prevalences not given)
Oxhoj (1979)	Yes	Yes	Probably	Yes	"Clerks who never welded"	Yes	Yes	UNSP	Yes
McMillan (1979)	Yes	Yes	Yes	Yes	UNSP	Yes	Yes	Yes	Yes
Akbarkhanzaden (1980)	Yes	No: age UNSP	Yes	Yes	UNSP	Yes	Yes	UNSP	Yes
Keimig (1983)	Yes	Yes	Yes	Yes	Yes (Self-administered)	Yes	Yes	UNSP	Yes
Hayden (1984)	No (Volunteers)	Yes	Yes	Yes	UNSP	Yes	Yes	UNSP	Yes
McMillan (1984)	No (Volunteers)	Yes	Yes	Yes	Yes	Yes	Yes	UNSP	Yes

S.E.S = Socioeconomic Status
UNSP = Unspecified

Table 1

Contaminants from Welding Operations

Contaminant	Source
Metal fumes	
Iron	Parent iron or steel metal, electrode
Chromium	Stainless steel, electrode, plating chromate primed metal
Nickel	Stainless steel, nickel-clad steel
Zinc	Galvanized or zinc primed steel
Copper	Coating on filler wire, sheaths on air-carbon arc gouging, electrodes nonferrous alloys
Vanadium, manganese, and molybdenum	Welding rod, alloys in steel
Tin	Tin coated steel
Cadmium	Plating
Lead	Lead paint, electrode coating
Fluorides	Flux on electrodes
Gases and vapors	
Carbon monoxide	CO_2 shielded, GMA, carbon arc gouging, oxy-gas
Ozone	GTA, GMA, carbon arc gouging
Nitrogen dioxide	GTA, GMA, all flame processes

(from Burgess, 1981)

Ranking of Harmful Agents

Contaminants of High Concern Priorty	Contaminants of Intermediate Concern Priority	
Chromium	Fluorides	Zinc
Nickel	Manganese	Copper
Ozone	Phosgene	Carbon monoxide + Carbon Dioxide
Oxides of Nitrogen	Iron	
Barium		

(from Tinkler and Graville, 1983).

Table 2

Summary of Chronic Respiratory Symptoms

Study (Yr)	Worksite (Prevalence of Siderosis/ Pneumoconiosis)	Statistically Significant Excess in Welders	Symptom	Smokers (or *Combined) RR	Smokers (or *Combined) P	Non-Smokers RR	Non-Smokers P
Hunnicutt (1964)	shipbuilding (34%)	yes	cough	1.7	0.05 <p<0.1	7.0	.05<p<.1
			sputum	1.7	NS	2.0	.05<p<.1
			wheezing	5.7	0.01	-	-
			dyspnea	4.3	0.05<p<0.1	∞	.05<p<.1
Kleinfeld (1969)	manufacturing (32%)	no	-	-	-	-	-
Fogh (1969)	factories (3% abnormal X-rays)	no	-	-	-	-	-
Peters (1977)	shipyard (0%)	no	-	-	-	-	-
Ulrich (1974)		yes	cough	-	-	3.7	.009
			sputum	-	-	3.4	.017
			dyspnea	*2.0	<0.05	4.8	.05<p<.1
Antti-Poika (1977)	non-shipyard (0%)	no	cough	-	-	-	-
			dyspnea	-	-	-	-
		yes	simple chronic bronchitis (sputum)	1.7	<0.01	-	-

Table 2 (continued)

Study (Yr)	Worksite (Prevalence of Siderosis/ Pneumoconiosis)	Statistically Significant Excess in Welders	Symptom	Smokers (or *Combined) RR	Smokers (or *Combined) P		Non-Smokers RR	Non-Smokers P
Ross (1978)	shipbuilding engineering (7%)	yes (smokers only 50-59)	cough with sputum	not given	-		-	-
					<0.05		-	-
							-	-
Oxhoj (1979)	shipyard (not reported)	yes (in non-smokers)	Cough sputum x 3 months	5.7	0.05 <p<.1		3.8	0.009
			wheezing	1.6	NS		4.8	0.012
			dyspnea uphill	0.9	NS		4.1	0.004
			dyspnea on level	2.8	NS		∞	NS
McMillan (1979)	shipyard (0%)	no	-	-	-	-	-	-
Akbarkhan-zadeh (1980)	shipyard (not reported)	yes	cough	1.5	NS	-	0.8	NS
			sputum	0.9	NS	-	3.4	0.05<p<.1
			persistent cough & sputum	1.6	NS	-	5.9	0.035
			chronic bronchitis	∞	0.0006	-	-	0.05<p<.1

Table 2 (continued)

Study (Yr)	Worksite (Prevalence of Siderosis/ Pneumoconiosis)	Statistically Significant Excess in Welders	Symptom	Smokers (or *Combined) RR	Smokers (or *Combined) P	Non-Smokers RR	Non-Smokers P
Keimig (1983)	manufacturing (not reported)	yes (in non-smokers)	sputum	-	NS	3.7	<0.05
			episodes of cough & sputum	-	NS		<0.05
Hayden (1984)	automotive products factories (2-12%)	yes (in smokers)	phlegm	not reported	<0.05	not reported	NS
McMillan (1984)	shipyard (3% 1/1 or more) (4% 1/0 or more)	yes	dyspnea	2.6	<0.05	∞	<0.05
				6 (in ex-smokers)	<0.05		
					-		

RR = Rate ratio (prevalence ratio)
NS = Not significant

Table 3

Summary of Pulmonary Function Findings
in Cross-Sectional Studies

Comparison of Means

Study	Significantly lower in welders	Smokers	Non-Smokers
Hunnicutt (1964)	Yes	FEV_1 **	No
		MEFR **	No
		MMEF **	No
Stanescu (1967)[a]	Yes	Static + dynamic compliance **	
	No	FEV_1, FEV_1/FVC	
Kleinfeld (1969)[a]	No		
Fogh (1969)[a]	No		
Peters (1973)[a]	No	(with internal controls)	
	Yes	(with external controls)	
		FEV ***	
		FEV_1 ***	
		PEFR *	
Antti-Poika (1977)	No		
Oxhoj (1979)	No	VC, FEV_1, MMEF, MEF_{50}, MEF_{25}	
	Yes	—	CV**) non- CC**) and ex TLC*) smokers
Akbarkhanzadeh (1980)	Yes		FEV_1
Keimig (1983)	Yes[b]	FEV_{25-75} FEF_{25} FEF_{50} FEF_{75}	FEF_{25-75} FEF_{25} FEF_{50} FEF_{75}
Hayden (1984)	No	FEV_1, PEFR	—
	Yes	MEF_{25}** MEF_{50}*	—
McMillan (1984)	No		

Comparison of Prevalance of Abnormality

Study	Excess in Welders	Smokers	Non-Smokers
Hunnicutt (1964)	Yes (obstruction)	41% vs 10%	28% vs 10%
McMillan (1984)	Yes	Restriction(*)[a]	Obstruction** (non-smokers)

(*) .05 <p<.1 * <.05 ** <.01 *** <.005 **** <.001

a combined analysis (smokers and non-smokers)

b all lower in welders (smokers and non-smokers) but not statistically significantly different from controls.

Table 4

Summary of Data on Chromium Excretion and Parameters of Renal Tubular Function in Welders and Chromium-platers (from Mutti et al., 1979)

Group (duration of exposure, yr)	Urine Chromium µg Cr/g creatinine	Chromium clearance (mL/min)	Number of Subjects with Abnormal Tests					
			B-glucuronidase		Proteinuria		Lysozymuria	
			n	%	n	%	n	%
A. (n=39) (4.5 ± 3.2)	5.3 ± 3.7	-	0/39	0	0/39	0	0/39	0
B. (n=36) (1.0 ± 0.4)	33.3 ± 6.9	8.8 ± 3.8	8/36	22	3/30	10	0/9	0
C. (n=24) (8.9 ± 7.3)	24.5 ± 12.5	10.3 ± 5.1	9/24	37	4/24	17	1/24	4

Groups A, B and C defined in text.

Table 5

Summary of Studies of Non-respiratory Cancer in Welders

Study (Yr)	Type	Site	No. of Cases	Relative Risk
Milham (1976)	PMR	Bladder	12	1.6
Howe et al. (1980)	Case Control	Bladder	17	2.8
Milham (1983)	PMR	Kidney	20	1.8
Olsen et al. (1984)	Case Control	Larynx	42	1.6
Hernberg et al. (1983)	Case Control	Nasal/ Sinonasal	21	2.8

Table 6

Summary of Lung Cancer Studies in Welders Based on Occupation Recorded on Death Certificates and/or Census Data

Study (Yr)	Method	No. of Cases	Relative Risk	
Registrar General (1978) (England & Wales)	SMR	246	1.51	
*Menck & Henderson (1976) (Los Angeles)	SMR	48	1.37	
**Sjogren et al. (1982) (Sweden)	SIR	96	1.44	p<0.001
Gottlieb (1980) (Louisiana)	Case Control	8	4.01	
Milham (1983) (Washington State)	PMR	153	1.35	p<0.01

* Mortality (1968-1970) plus incidence (1972-1973).

** SIR = Standard Incidence Ratio

Table 7

Summary of Lung Cancer Studies in Welders

Study Year	Source of Population	Exposure Data	No.of Cases	Relative Risk (RR)	
Shipyard Welders					
1. Cohort Studies					
Beaumont (1981)	Seattle Union	No	50	1.32	($p=0.06$[a])
Putoni (1979)	Genoa Shipyards	No	7	1.32	
2. Case-control Studies					
Blot (1980)	Coastal Virginia	No	11	1.3[b]	
Blot (1978)	Coastal Georgia	No	11	0.7	
Non-shipyard Welders					
1. Cohort Studies					
Dunn (1968)	California Unions	No	49	1.05	
Polednak (1981)	Oak Ridge Facilities	Yes	17	1.50	
Redmond (1979	Pennyslvania Steel Companies	No	14	1.51	($p<0.05$)[c]
Sjogren (1980)	Swedish Companies (stainless steel)	Yes	3	4.41	($p=0.03$)
Ott (1976)	Michigan Chemical	No	2	1	(approx)
Becker (1985)	German Factories	No	6	0.95[d]	
2. Case-control Studies					
Breslow (1954)	California Hospitals	No	14	7.2	
Decoufle (1978)	New York Hospitals	No	9	0.9	
Gerin (1984)	Montreal Hospitals	No	12	2.8	($p<0.05$)[e]

a 20 year latency: RR 1.74 ($p<0.001$)

b When restricted to shipyard workers, RR was 1.04

c In comparison to other steel workers, RR was 1.13

d On internal comparison to turners at same factories, rate ratio for lung cancer was 1.7 (95% confidence interval 0.7-4.0)

e 95% confidence interval 1.4-5.8